Use of antimicrobial coated polyglactin sutures versus plain polyglactin sutures in oral malignancies

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A dissertation submitted to the M.G.R. Medical University, Tamil Nadu: in partial fulfillment of the requirement for the M.S. Branch I(General Surgery) examination held in April 2016.

CERTIFICATE

This is to certify that the dissertation titled 'Use of antimicrobial coated polyglactin sutures versus plain polyglactin sutures in oral malignancies' is an original bonafide work by Dr. Abhilasha Gloria Singh, post graduate resident in Masters of General Surgery 2013-2016 at the Christian Medical College, Vellore, towards partial fulfillment for the MS General Surgery Branch I final examination held in April 2016.

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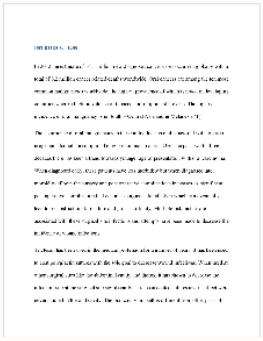

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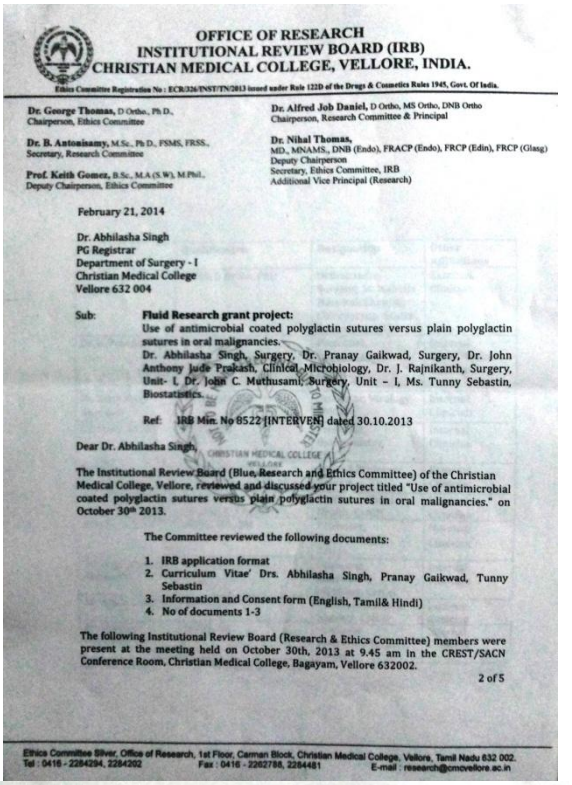

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February 21, 2014

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Sub: **Fluid Research grant project:**
Use of antimicrobial coated polyglactin sutures versus plain polyglactin sutures in oral malignancies.
Dr. Abhilasha Singh, Surgery, Dr. Pranay Gaikwad, Surgery, Dr. John Anthony Jude Prakash, Clinical Microbiology, Dr. J. Rajnikanth, Surgery, Unit- I, Dr. John C. Muthusami, Surgery, Unit – I, Ms. Tunny Sebastin, Biostatistics.

Ref: IRB Min. No 8522 [INTERVEN] dated 30.10.2013

Dear Dr. Abhilasha Singh,

The Institutional Review Board (Blue, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project titled "Use of antimicrobial coated polyglactin sutures versus plain polyglactin sutures in oral malignancies." on October 30th 2013.

The Committee reviewed the following documents:

1. IRB application format
2. Curriculum Vitae' Drs. Abhilasha Singh, Pranay Gaikwad, Tunny Sebastin
3. Information and Consent form (English, Tamil& Hindi)
4. No of documents 1-3

The following Institutional Review Board (Research & Ethics Committee) members were present at the meeting held on October 30th, 2013 at 9.45 am in the CREST/SACN Conference Room, Christian Medical College, Bagayam, Vellore 632002.

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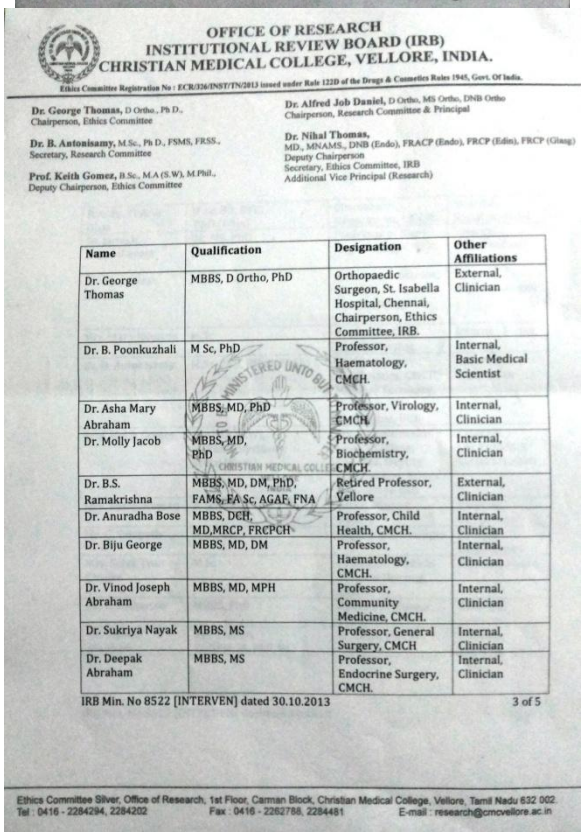

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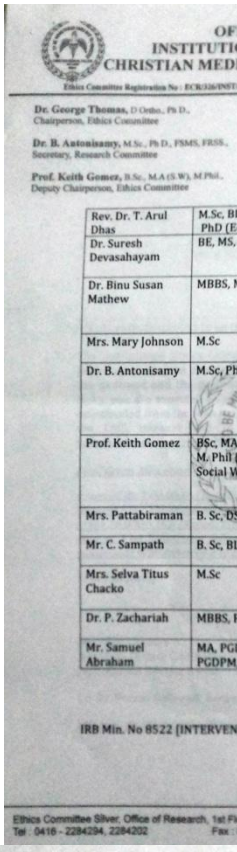

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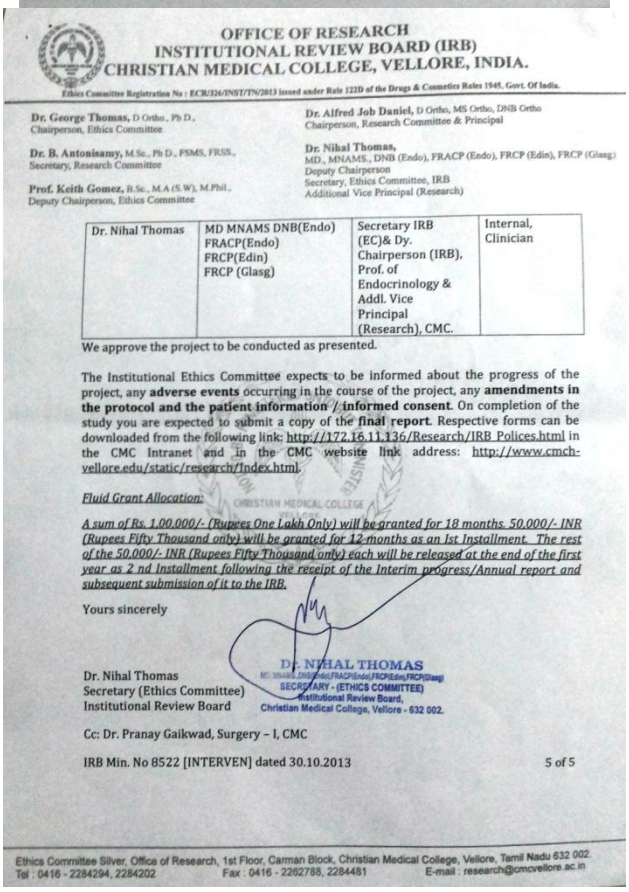

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We approve the project to be conducted as presented.

The Institutional Ethics Committee expects to be informed about the progress of the project, any **adverse events** occurring in the course of the project, any **amendments in the protocol and the patient information / informed consent**. On completion of the study you are expected to submit a copy of the **final report**. Respective forms can be downloaded from the following link: http://172.16.11.136/Research/IRB_Polices.html in the CMC Intranet and in the CMC website link address: http://www.cmch-vellore.edu/static/research/Index.html.

Fluid Grant Allocation:

A sum of Rs. 1,00,000/- (Rupees One Lakh Only) will be granted for 18 months. 50,000/- INR (Rupees Fifty Thousand only) will be granted for 12 months as an 1st Installment. The rest of the 50,000/- INR (Rupees Fifty Thousand only) each will be released at the end of the first year as 2 nd Installment following the receipt of the Interim progress/Annual report and subsequent submission of it to the IRB.

Yours sincerely

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SECRETARY - (ETHICS COMMITTEE)
Institutional Review Board,
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Dr. Nihal Thomas
Secretary (Ethics Committee)
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Cc: Dr. Pranay Gaikwad, Surgery – I, CMC

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ACKNOWLEDGEMENT

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I thank my guide **Dr. Pranay Gaikwad**, Professor, Dept. of Surgery, CMC Vellore for his continuous and valuable support and guidance.

I also thank by co-investigators **Dr. John.C. Muthusami** Professor, Department of Surgery, **Dr. Rajnikanth**, Professor, Department of Surgery and for their help and advice.

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I express my sincere gratitude to all my teachers and colleagues in the Department of General Surgery for their support and encouragement.

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Above all, I thank God for his grace and guidance.

INTRODUCTION

In 2012, an estimate of 14.1 million new diagnosed cancer cases occurred globally with a total of 8.2 million cancer related deaths worldwide. Oral cancers are among the ten most common malignancies worldwide, the highest prevalence of which is noted in developing countries where a high prevalence of tobacco consumption still exists. The highest incidence of oral malignancy is in South – Central Asia and in Melanesia (1).

The occurrence of oral malignancies in these individuals is multi-factorial with tobacco usage and alcohol consumption being predominant causes. Over the past two to three decades there has been a trend towards younger age of presentation with oral carcinoma. When diagnosed early, these patients have less morbidity but when diagnosed late, morbidity of both the surgery and post operative complications increases. A significant post operative complication that occurs is surgical site infection which can eventually lead to reconstruction flap failure and gross morbidity. Multiple risk factors are associated with these surgical site infections and attempts have been made to decrease the incidence of wound infections.

Triclosan has been used in the medical profession for a number of years. It has been used to coat polyglactin sutures with the sole goal to decrease wound infections. When used at other surgical sites like the abdominal cavity and thorax, it has shown to decrease the infection rate at the surgical site

significantly. Triclosan coated sutures and its effect were never studied in the oral cavity. The oral cavity in itself is different from other parts of the body and so are the wounds. Microbial colonization of the oral cavity in the presence of a malignancy has never been studied. Whether it is the same as that in a normal oral cavity is not known. Only a thorough understanding of the colonization can effectively lead to reduction in these infections that cause gross morbidity to patients who undergo surgical procedures.

This study aims to study the infection rates when this suture (triclosan coated polyglactin 910 suture) is used within the oral cavity following oral malignancy surgeries. This study also aims to understand further the oral microbiology that exists in the oral cavity of those who have a pre-existing malignancy with microbiological analysis of post operative wound infections in these patients.

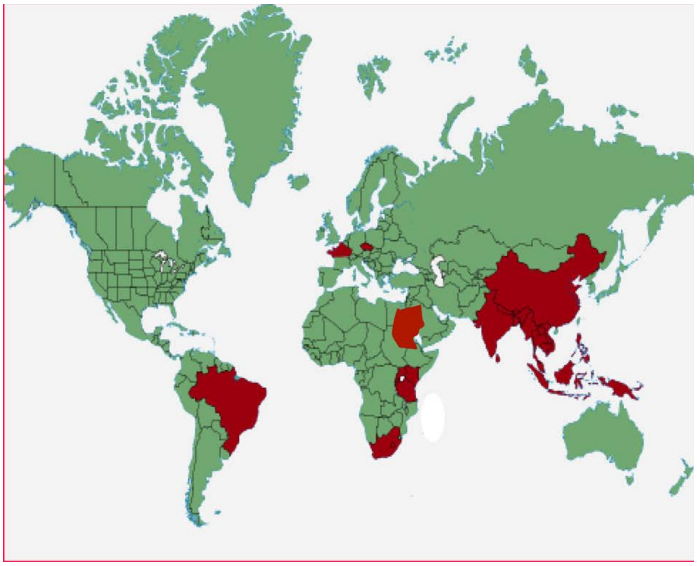
OBJECTIVES

1. The primary objective is to compare reduction of wound infection rate in Triclosan coated polyglactin 910 sutures as compared to plain coated polyglactin sutures in patients with oral malignancy treated in the unit over the past one year.

2. The secondary objective is to include a microbiological study of all these patients in relation to wound infection.

LITERATURE REVIEW

Cancer is responsible for causing more deaths in present age than cerebrovascular accidents and coronary artery diseases. The global cancer burden is progressively increasing with the WHO estimating 20 million new cases of cancers by the year 2025. Oral malignancies are among the top ten most common malignancies in the world with the highest prevalence being in the South – Central Asian and Melanesia population. In 2012, 2.1% of all cancers globally were malignancies involving the lips and the oral cavity. These malignancies were more common in men with a incidence of 22.9 per 100000 in men and 16.9 per 100000 in women in areas of high incidence namely Melanesia. There were a total of 145000 deaths as a direct result of which 77% occurred in less developed countries with a lower economical status(1).



In India, there is an average of 100,000 new cases of oral cancer each year with Bhopal reporting the highest incidence with 10.9 per 100,000 and 9.6 per 100,000 for tongue and mouth cancer respectively(2). Head and neck cancers contribute to a substantial mortality in South Asian region. In 2010, the estimated welfare and economically loss was estimated to be USD 16.9 billion(3). The 5 year survival rate for advanced oral cancer is 20% (4) with the causes of death usually being loco-regional recurrence or distant metastasis, early diagnosis and detection of oral cancers can bring down the mortality rate significantly as the 5 year survival for early oral cancer is 80% (5).

Anatomy and physiology of the oral cavity:

The oral cavity is formed by the lips, tongue, the cheeks and the floor of the mouth. It is bounded anteriorly by the vermilion border of the lips, inferiorly by the circumvallate papilla on the posterior third of the tongue. The superior border is at the junction between the hard and the soft palate with the lateral borders being formed by the anterior tonsillar pillars(6)(7).

The embryological development of the head and the neck starts in the 4^{th} to 5^{th} week of development from the 1^{st} and the 2^{nd} pharyngeal arches. The center of the face is formed by the stomodeum around which exists the first pharyngeal pouch. Each pharyngeal pouch contains three components – epithelial

endoderm, core of mesenchymal tissue and the surface ectoderm. The mesenchymal tissue is responsible for the formation of the muscular components of the face, while the neural crest cells within the mesenchymal core forming the skeletonal structures of the pharyngeal arch. Around the stomodeum by 42 weeks, develop caudially the maxillary prominences, laterally the maxillary and cranially the frontonasal prominences, which further undergo differentiation to lead to formation of the face(8).

PROMINENCE	STRUCTURES FORMED
Frontonasal	Medial and lateral nasal prominence, forehead, bridge of nose.
Maxillary	Lateral portion of the upper lip, cheeks
Medial Nasal	Tip of the nose, crest, philtrum of the upper lip
Lateral Nasal	Nasal alar
Mandibular	Lower lip

FIRST PHARYNGEAL ARCH – MANDIBULAR

Nerve – maxillary and mandibular branch of the Trigeminal nerve

Muscles – Forms the mylohyoid, tensor palpatine, tensor tympani and the anterior belly of the digastrics and all the muscles responsible for mastication namely – medial and lateral pterygoids, temporalis and masseter.

Skeletonal component – It forms the mandible, malleus, incus, sphenomandibular ligament, anterior ligament of malleus, premaxilla, maxilla, zygomatic bone and a portion of the temporal bone.

SECOND PHARYNGEAL ARCH – HYOID:

Nerve – Forms the facial nerve

Muscles – The mesenchymal core forms the posterior belly of the digastrics, stylohyoid, stapedius and all the muscles of facial expression namely – the orbicularis oculi, orbicularis oris, platysma, buccinators, auricularis and the frontalis.

Skeletonal component – It forms the lesser horn and the upper half portion of the hyoid bone, stylohyoid ligament, styloid process and the stapes.

The tongue starts developes at around the 4^{th} week from two lateral lingual swellings and one medial swelling which is called the tuberculum impar. The copula or the hypobrachial prominence forms a second median swelling which is formed from the second, third and fourth arches. The posterior part of the fourth arch marks the origin of the third median swelling which forms the epiglottis. The lateral lingual swellings overtake the growth of the first medial swelling to form the anterior 2/3rds of the tongue. The anterior 2/3rds of the tongue is covered by mucosa derived from the first pharyngeal arch – the

mandibular branch of the trigeminal nerve. The posterior third of the tongue derive its mucosa from the fourth pharyngeal arch(8).

The primary palate develops when the two medial nasal prominences fuse in the midline and forms the intermaxillary segment. The secondary palate is formed by fusion of the palate plates which arise from the maxillary process. This segment forms the primary palate when it grows posteriorly and fuses with the secondary palate at the incisive foramen.

Histologically, the entire oral cavity is lined by thick stratified squamous epithelial with the lamina propria acting as a supporting layer(9). The freely mobile mucosal lined surfaces namely – the floor of the mouth, underside of the tongue, cheeks and lips are lined by non keratinized squamous epithelial. Epithelium over the gingivae, hard palapte and the uppersurface of the tongue are highly keratinized. Below the epithelial lining there are thick collagenous submucosal layer which contains accessory salivary glands. The submucosal layer over the bone is thin.

Anatomically the entire oral cavity can be divided into seven sub-divisions as below(10):

- Anterior two-thirds of the tongue
- Retromolar trigone
- Alveolar sulcus

- Lips
- Buccal mucosa
- Hard palate
- Floor of the mouth

This traditional grouping has not been productive either for clinical medicine with regard to giving insight regarding etiological factors or techniques for examining the mouth.

A rough rule of thumb exists for location of oral cancer which is as follows – one quarter occur in the most anterior portion of the floor of the mouth, one quarter occur in each of the gingivobuccal sulcus and the last quarter in the other locations of the mouth. This forms a horseshoe area in the oral cavity where occurs the highest predominance of cancerous oral lesions(11).

Majority of saliva produced is from the major salivary glands namely the submandibular, parotid and the sublingual glands. The saliva once released from the glands forms a thin film like layer over the entire oral cavity. Besides lubricating the oral mucosa and protecting it from abrasive lesions, it also has antibacterial and antifungal properties. It prevents dental caries formation, attrition, and dental erosions(12). IgG, IgA and IgM antibodies are found abundantly in saliva and contribute to local oral immunity(13). Lederman in his paper titled the anatomy of cancer refers to two bulbous areas on either side of the tongue, known as the Lederman's oral mucus reservoirs. This oral mucus

reservoir are bounded by the gingivobuccal sulcus on either side, posteriorly by the tonsillar pillars, the medial border being formed by the posterior part of the tongue and is continous anteriorly with the floor of the mouth. There is stagnation of saliva in the gingivobuccal sulcus and the floor of the mouth which act as the reservoir and the gutter system in the oral cavity. This area is also responsible for pooling of saliva in an upright individual. This reservoir includes the horseshoe area where the majority of oral malignancies occur most commonly within the oral cavity(11)(14).

Microbiology:

The oral cavity has a diversion bacterial flora, it is important to understand the normal microflora in the oral cavity prior to understanding the flora in a diseased oral cavity. To our knowledge, there is preferential colonization of sites in the oral cavity with site specific adhesions on the bacterial surface, which enables it to bind to specific sites on the oral surface(15). A study conducted by Aas et al to define the bacterial growth from nine different sites in the oral cavities of normal healthy adults revealed the following six phyla(16):

- *Firmicutes* : *Streptococcus, Selenomonas, Gemella, Veillonella, Eubacterium*
- *Actinobacteria: Actinomyces, Rothia*
- *Proteobacteria: Neisseria, Campylobacter*

- *Bacteroidetes*
- *Fusobacteria: Fusobacterium, Leptotrichia*
- *TM7*

These were broadly divided into Gram positive and gram negative organisms, then further into anaerobic and aerobic organisms.

The organisms that were cultured from the surgical sites in the oral cavity after head and neck cancer surgeries were polymicrobial and included *Staphylococcus aureus* (17), *Pseudomonas aeruginosa*, *actineobacter* (18), *Escherichia coli, Klebsiella, non hemolytic Streptococcus, coagulase negative staphylococcus* (19) to name a few.

Premalignant conditions of the oral cavity:

In 1805, suggestions were given to a European panel, that there are a group of benign diseases that will if followed for a prolonged duration will lead to invasive malignancy. This was the beginning of the concept of pre-cancer. In 1870s, Sir James Paget described “smokers patch” also commonly now known as leucokeratosis(20). The World Health Organisation defined them as potentially malignant disorders which defined to two groups:

- Any tissue that has been morphologically altered such that it is at higher risk of becoming malignant than its otherwise normal counterpart is called a precancerous lesion.
- Precancerous condition is a state that is associated with a higher risk of malignancy

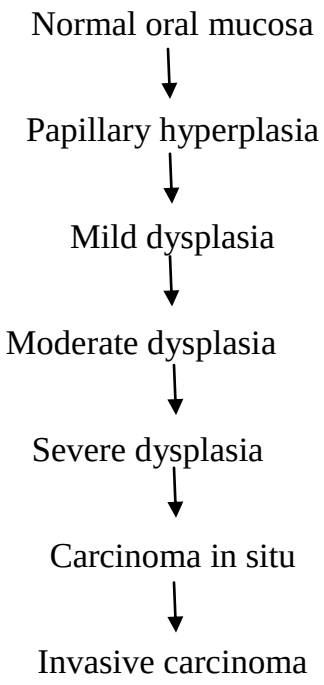
These premalignant lesions inspite of their clinical appearance are diagnosed only based on histology. The limitation occurs that even histology will only provide insight into whether the lesion has malignant potential and never predict malignant transformation(21). The risk of transformation into malignancy from a precancerous lesion has been reported between 6.6% to 36.4%.(22)

Potentially Malignant Disorders (PMDs) (23)

Premalignant lesions	Premalignant conditions
Leukoplakia	Lichen planus
Erythroplakia	Discoid lupus erythematosus
Proliferative verrucous leukoplakia(PVL)	Epidermolysis bullosa
Viadent leukoplakia	Verruciform xanthoma
Candida leukoplakia	Graft-versus-host-disease
Reverse smokings' palate	Cheilitis glandularis
Verrucous hyperplasia	Xeroderma pigmentosum
Oral verrucous carcinoma	Syphilis (third stage)
Dyskeratosis congenita	Plummer-Vinson syndrome
Actinic cheilosis	Malnutrition
Keratoacanthoma	Vitamin A, B, C deficiency
Oral submucous fibrosis	Immunosuppressive diseases [AIDS]

Carcinogenesis:

The mechanism of carcinogenesis in the oral cavity is a multifocal highly complex process. It occurs when several genetic alterations occur on the squamous epithelium.



Field cancerization has been noted in tissues lined by either squamous epithelium as in the oral cavity or transitional epithelial as in the urinary bladder. It refers to the ability of cancer to develop at multiple sites. Over the years, there are various sites of malignant transformation within the oral cavity. Mutations in the tumour suppressor p53 genes have been noted in areas of

premalignant lesions and in the foci of carcinoma(24). Dysregulation of miRNA which are classified as proto-oncogenes are also involved in inhibition of differentiation, causes uncontrolled cell proliferation, and induces invasive behavior within cells and its progression to oral cancer(25).

Smoking, alcohol and other exogenous factors over prolong durations of exposure can cause mutational expressions and multifocal presentations of tumor suppressor genes. These mutational adaptations may also change the level of resistance to therapy.

Risk factors:

There are multiple risk factors that all contribute to the development of oral malignancy. These factors may either be independent risk factors or may be additive to increasing the risk of development of oral cancer.

Alcohol consumption has been associated with an increase in the risk of squamous cell carcinomas of the upper aerodigestive tract and also an increase in the risk of adenocarcinomas in the pancreas, distal stomach and colon – the later association being not as strong as the former. A study conducted by Thomas et al showed that there with increasing amounts of alcohol consumption there was an increased risk of malignancy. There was also an

increase in both pharyngeal and oral cancers in regular and heavy consumers of alcohol(26)(27).

The risk of oropharyngeal cancers increases with duration of smoking cigarettes, number of cigarettes smoked per day in addition to the manner in which it is smoked. If smoked in the form of unfiltered cigarettes or in the form of cigars there is a further increased risk as when compared to smoking of filtered cigarettes(28). The smoking of one cigar is equivalent to smoking an entire pack of unfiltered cigarettes(29). In individuals who have quit smoking cigarettes for 10 years or more, the cessation has been associated with a sharp decrease in the risk of cancer. The reduction in the relative risk of development of oral and pharyngeal cancer suggests that smoking may play a role in the late stage of oropharyngeal carcinogenesis.

The action of alcohol and smoking on the overall risk appear to be greater than additive and is more a multiplicative effect. These two carcinogens exhibit a biological synergism, where alcohol potentiates the action of the carcinogens in the cigarette smoke. There a 35-fold increase among the individuals who consumed more than four alcoholic drinks per day and two or more packs of cigarettes(28). Inspite of this biological synergism, it is evident that in the absence of smoking, alcohol independently increases the risk or pharyngeal and oral malignancy.

Tobacco usage and the chewing of betel quid has been in found in association with alcohol consumption to all contribute to the development of oral squamous cell carcinoma. There has also been a statistically significant association with betel nut chewing and oral cancer, the risk of which is increased if the betel nut quid is kept in the oral cavity and juices swallowed. A study conducted by Ko YC et al in Taiwan concluded that in the presence of all these three – smoking, alcohol consumption and betel nut chewing, there was a 123-fold increase in the incidence of oral cancer(30).

Oral hygiene, dentition, jagged teeth, decayed teeth have been thought to increase the risk of development of oral malignancy. In a case-control study conducted by Talamini et al, there was evidence that a poor general oral hygiene (dental caries and tartar) had a 4.5 fold more in cases than among controls even after giving allowance for factors such as smoking, drinking , fruit and vegetable consumption(31). Another case-control study conducted by Zheng et al, noted that in men who had reportedly never brushed their teeth an increase in the risk of oral malignancy by 7-fold. These studies also confirmed that oral hygiene is an independent risk factor for development of oral cancer(32).

Numerous studies preformed have shown a protective effect of high fruit diet with a reduction in oral cancer risk of 20-80%(33). The consumption of certain spicy food and hot beverages that are specific to certain cultures and indigenous

areas have shown to increase the overall risk of oral malignancy. In a case-control study conducted in India by Notani et al, the use of red chilli powder was an risk factor for cancers of the aerodigestive system. It also emerged that consuming hot beverages increased the risk of oesophageal and pharyngeal cancers(34). In Brazil, an indigenous variety of tea, chimarrao and mate has been associated with an increase in the risk of only tongue cancer(35), there was no increase risk with consumption of tea or coffee. Notani et al also showed the protective effect of consumption of fish, vegetables, buttermilk and pulses. These factors were risk modifiers to those who chewed or smoked tobacco.

The human papillomavirus (HPV) has been found to be associated with the progression to oral malignancies. HPV positive squamous cell carcinomas are characteristically different from HPV negative head and neck cancers with respect to genetic alterations, clinical progression and therapeutic response(36). A study performed at the University of Iowa, aimed at looking at whether HPV found in the oral exfoliate cells of the oral cavity was an individual risk factor for the development of oral malignancies. They found that in those infected by the oncogenic HPV or HPV-HR (most commonly HPV16) was independent of tobacco and alcohol use(37) but did act synergistically with alcohol(38). Their conclusion was that HPV testing could thus be predictive of HPV related head and neck cancers. A systemic review and meta-analysis done by Ndiaye et al,

there was prevalence of 24.2% of HPV-DNA in the oral cavity. In view of this high prevalence especially of HPV16, there may be benefit of prophylactic vaccinations(39).

Staging of the disease:

Once the diagnosis of an oral malignancy has been made, evaluation for locoregional and systemic spread are carried out following which the disease is clinically staged. Imaging modalities like magnetic resonance imaging, computed tomography can be applied when the disease is more advanced and will provide a more accurate T (tumour) and N (nodal) staging. Whenever clinical findings are unclear or uncertain, appropriate imaging modalities is to be utilized. The nodal status for all patients must be evaluated thoroughly in the pretreatment phase. It is also to be noted that nodes more than 3cm are not to be considered as single nodes but as a confluence of multiple nodes or as tumour within the soft tissues of the neck itself.

The most common sites of metastasis in head and neck malignancies are the lungs and the bone with hepatic and brain metastasis been less frequent. Mediastinal nodes are considered distant metastasis. Metastatic workup in the pretreatment phase is crucial in cases of advanced disease(40)(41).

TNM staging system for cancers of the lips and oral cavity

Primary tumor (T)

Tx	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor ≤ 2 cm in greatest dimension
T2	Tumor > 2 cm but ≤ 4 cm in greatest dimension
T3	Tumor > 4 cm in greatest dimension
T4	(lip) Tumor invades through cortical bone, inferior alveolar nerve, floor of mouth, or skin of face, ie, chin or nose[a]
T4a	(oral cavity) Tumor invades adjacent structures (eg, through cortical bone, into deep [extrinsic] muscle of the tongue, maxillary sinus, or skin of face); resectable lesions
T4b	Tumor involves masticator space, pterygoid plates, or skull base and/or encases internal carotid artery; unresectable lesions

Regional lymph nodes (N)

Nx	Regional nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single ipsilateral lymph node, ≤ 3 cm in greatest dimension
N2	Metastasis in a single ipsilateral lymph node, > 3 cm ≤ 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none > 6 cm in greatest dimension; or in bilateral or contralateral lymph nodes, none > 6 cm in greatest dimension
N2a	Metastasis in a single ipsilateral lymph node, > 3 cm but ≤ 6 cm in greatest dimension
N2b	Metastasis in multiple ipsilateral lymph nodes, none > 6 cm in greatest dimension
N2c	Metastasis in bilateral or contralateral lymph nodes, none > 6 cm in greatest dimension
N3	Metastasis in a lymph node, > 6 cm in greatest dimension

Distant metastases (M)

Mx	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Stage grouping

Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1	N1	M0
	T2	N1	M0
	T3	N1	M0
Stage IVA	T4a	N0	M0
	T4a	N1	M0
	T1	N2	M0
	T2	N2	M0
	T3	N2	M0
	T4a	N2	M0
Stage IVB	Any T	N3	M0
	T4b	Any N	M0
Stage IVC	Any T	Any N	M1

From Greene FL, Page DL, Fleming ID, et al (eds): AJCC Cancer Staging Manual, 6th ed. New York, Springer-Verlag, 2002.

[a] Superficial erosion alone of bone/tooth socket by gingival primary is not sufficient to classify a tumor as T4.

For patients with early disease with Stage I and II, the 5 year survival is as high as 80%. This drops as the disease becomes more advance with Stage III and IV having a 5 year survival of 40%(42).

Management:

Oral malignancy if diagnosed early is completely treatable. The primary goal of treatment is to cure the disease, preserving functionality is also of avid importance. Nerves are the most important structure to preserve if functionality is to be preserved. In early head and neck malignancies, there is a single modality of treatment namely either surgery or radiation therapy, ant the outcomes are both comparable. More advanced cases are discussed with a

multidisciplinary team so as to attain individualized patient treatment prior to making any treatment plans.

Currently there are three modalities of treatment:

- Surgical
- Radiation therapy
- Chemotherapy

1. Surgery:

   In oral cavity tumors the goal of treatment is maximum tumour resection while being able to maintain functionality. The main treatment modality is surgical resection. To ensure an adequate surgical resection one must attain a resection margin of atleast 1-1.5cm. For early lesions, the defect closure can be done in multiple ways:

   Primary closure

   Split-thickness skin graft

   Temporoparietal fascial flap

   For larger defects, following resections – glossectomy (total/partial), mandibulectomy(hemi/marginal) primary closure is not feasible.

   Reconstruction methods employed include:

   Rotational flaps:

   Nasolabial flap

Facial artery musculomucosal flap (FAMM)

Submental artery island flap

Forehead flap

Deltopectoral flap

Pedicled flaps:

Pectoralis major myocutaneous flap

Latissmus dorsi myocutaneous flap

Free flap reconstructions:

Radial forearm free flap

Fibula osseocutaneous free flap

If multiple nodes are involved, large nodes with extracapsular extension, then neck dissections are performed. Based on the number of structures spared and the nodal levels cleared, three are characterized, namely:

- Radical neck dissection: Level I – V nodes cleared

  Spinal accessory nerve scarificed

  Sternocleidomastoid removed

  Internal jugular vein scarificed

- Modified radical neck dissection: Level I – V cleared.

  Spinal accessory, sternocleidomastoid and internal jugular vein all spared.

- Selective neck dissection: In oral malignancy, this is representative of supraomohyoid neck dissection, which consists of clearance of

cervical lymph nodes in level I-III while preserving the internal jugular vein, sternocleidomastoid and the spinal accessory nerve.

2. Radiation Therapy:

   Radiation therapy may be administered as external beam radiation, brachytherapy (primary interstitial brachytherapy), intensity modulated radiation therapy (IMRT). Radiation though can be given as primary treatment for oral malignancy is only rarely given so. It is usually reserved for post operative treatment in those patients who have a higher risk of local and regional recurrence. These include patients with a positive tumour margin, close resection margins (less than 1cm), large tumours (T3 or T4), perineural or perivascular invasion, tumour with greater than 4mm depth of invasion, nodal metastasis with extension beyond the capsule and multiple lymph nodal involvement. If any of these are present there is an indication for post operative radiation therapy with any of the modalities mentioned.

3. Chemotherapy:

   Over the past few years, chemotherapy has been playing a more important role in the management of head and neck malignancies. It is

currently being used in advanced cases, unresectable tumors or in tumor recurrences.

Wound infection:

There are limitations of surgery in head and neck cancers are related to the risk of peri-operative complications. In patients with oral malignancy, the occurrence of infection can lead to multiple difficulties including prolonged hospitalization, poor cosmetic outcomes, delay in initiation of adjuvant therapy and wastage of financial supports. In patients who underwent radical neck dissections as part of treatment for oral malignancy, infection rates without antimicrobial prophylaxis have been reported to be as high as 68%(43), which on giving antibiotic prophylaxis have decreased to 21%(17) – 41.8%(19).

The Center for Disease Control, has classified surgical wounds into four categories based on the degrees of contamination, degree of inflammation, whether the gastrointestinal system/respiratory or urogenital system has been opened.

There are classified into four categories(44):

1. Class I / Clean wounds:

   This is a completely uninfected operative wound, where there is no inflammation and the respiratory, gastrointestinal or uninfected urinary tract is not entered. These wounds are usually primarily closed. Skin incision wounds following blunt trauma are included in this category.

2. Class II / Clean – contaminated wounds:

   In these wounds, there is no major break in the sterile technique, it is done in a controlled manner. So any operative wound where the urogenital, gastrointestinal or respiratory system are opened without unexpected contamination. These include operations of the biliary tract, appendix, oropharynx and vagina.

3. Class III / Contaminated wounds:

   Wounds that are fresh, open accidental wounds, this includes operations where there is a breach in the sterile techniques or spillage from the gastrointestinal tract. Incisions where acute, non-purulent inflammation is noted also is classified in this category.

4. Class IV / Dirty infected wounds:

   Wounds secondary to trauma that contain the devitalized tissue and also involves ongoing clinical infection or perforation of viscera. This means that organisms that cause post operative infection were in the operating field at the initiation of the operation.

Oral surgery is conducted in a clean-contaminated surgical field, as the oral cavity is continuously bathed in oral secretions that are rich in microflora. The oral gutters allows for stagnation of saliva in the inferior alveolar ridges and at the floor of the oral cavity. In a study conducted looking at sites of wound infection following surgery for oral malignancy, the site for highest occurrence of infections were the gingiva of the lower alveolar ridge and the base of the tongue(18). The relationship between the stagnation of saliva and infection has not been studied and is still uncertain.

The Center for Disease Control have defined superficial surgical site infections as any infection that occurs within 30 days following surgery and must include one of the following:

- The incision site should have purulent discharge
- Aseptically obtained culture of the tissue or fluid from the incision site should contain organism isolates.

- There should be atleast one of the following – redness, warmth, tenderness, pain or localized swelling and the incision site is opened by the surgeon. This is not included if the culture attained are negative for isolates of organisms.
- If the surgeon or attending physician make the diagnosis of superficial surgical site infection.

It was also important that stitch abscesses and incisional surgical site infections that extended into the fascial or muscular planes were not included.

Causes for wound infection:

There are numerous factors that may lead to an increase in the incidence of wound infections post operatively. These risk factors included diabetes, poor nutritional status, prior chemotherapy or radiation therapy, dental status, preoperative hospital stay, stage of the disease, intra-operative blood transfusions, flap reconstructions and preoperative tracheostomies. The validity of each of these risk factors is questionable with various studies evaluating different factors.

A study conducted to evaluate for risk factors for infection following oral surgery revealed that the only patient dependent factor for infection was male sex(18). There were no other patient factors namely smoking, tobacco usage or

alcohol consumption that had any significant association with an increase in infection rates. Another study conducted by Cloke et al concluded that there was no statistical significance in infection rates with age, sex or consumption of alcohol or smoking cigarettes(17).

An increase in the risk of infection has been noted with various disease characteristics. These disease include the preoperative T (tumour) stage and the location of the primary tumour. The most frequent site of infection in oral malignancy occurred when the primary tumour was located at the lower alveolar ridge. The second most common site was the base of the tongue(18). Tumours that occurred primarily on the tongue had the lowest risk of infection. The initial T(tumour) staging of the tumour also plays an important role in the overall outcome of the patient. Patients with advanced tumours (T4) lesions had to undergo more extensive dissections which leads to a greater chance of development of wound infections. These extensive surgical resection also leave large defect that require a larger reconstruction which are thus more predisposed to wound infections(19). A few studies the significance of the increasing N (nodal) staging has been associated with an increase in the wound infection rate(45), Penel et al(19) and Belusic-Gobic et al(18) did not report similar findings.

The surgical procedure in itself has also shown to alter the rate of infections. In those patients who underwent reconstructive surgeries there was an increase in

the wound infection rate than those who underwent a primary wound closure(45)(19). The wound infection rate in those where reconstruction was done with either using a muscular flap or a myocutaneous flap ranged from 20 – 37%(46). The infection rates in those whose surgical sites were either closed primarily or a skin graft had infection rates of only 3-15%(18). The cause of this could be attributed to surgical errors either in flap construction or in hemostasis of the flap in itself. Among all the flap reconstructions, the pectoralis major myocutaneous flap has been associated with a higher incidence of wound infections(18). Ischemia and flap necrosis predispose to infections and maybe caused by multiple factors. These factors include inadequate hemostasis, poorly mobilized grafts that are under undue tension due to inadequate tunneling, vascular insufficiency or direct tissue trauma. Ensuring proper flap planning and reducing technical errors could contribute to decreasing the infection rate in these major reconstructive surgeries.

Intraoperative blood transfusions have been found to be associated with an increased risk of wound infection but whose statistically significance has not been proven. In the study conducted by Belusic-gobic et al, it was associated with 88.67% of wound infections(18)(47).

Two studies preformed looked at the preoperative platelet count and its association with risk of surgical site infections in head and neck malignancies.

Schwartz et al and Pelczar et al reported that thrombocytosis was associated with an increase in the risk of wound infection. Platelets of more than 300000 in Schwartz study and 400000 in Pelczar study were seen to be a statically significant cause for increase in the infection rate(48)(49).

The history of preoperative radiation therapy and the wound infection rates is conflicting. Girod et al conducted a multicenteric study with 159 patients undergoing aerodigestive head and neck cancer surgeons with 87% of cases being located in the oral cavity and the oropharyngeal cavity. In the study the history of previous radiation therapy was statistically significant unlike in other studies(19). Robbins et al studied 400 patients who underwent major head and neck tumour resections and found no association between wound complication and radiation(45).

Penel et al studied the risk factors for wound infection for 165 consecutive cases over the duration of 24 months(19). They reported that in patients who had received preoperative chemotherapy the risk of wound infection was 68% which was reduced to 37% in those who did not receive chemotherapy. This finding was consistent with the study conducted by Corey et al where he observed surgical complications with patients who were receiving chemotherapy(50).

There was an association of wound infection with those patients who had a longer duration of pre-operative stay or previous hospitalizations. This was also

linked to infection with resistant organisms – either intrinsic resistance or acquired antibiotic resistant organisms. (51)

In most studies there was no association found between the dental status of a patient and the risk of wound infection following surgery. One prospective study of 186 head and neck malignancy patients conducted by Chaukar et al at the Tata memorial center reported that a significant factor associated with wound infection was oral hygiene and that preoperative scaling and good oral hygiene practices are to be considered for reduction in wound complications(52).

High risk of wound infections following head and neck surgeries thus is an adequate indication for antibiotic prophylaxis in the perioperative period. It is now routine to adminster antibiotics for atleast 48 hours in the post operative period and this has shown to decrease infections from 78 to 33%(53). The current recommendation is to adminster one of the following(54):

- Cefazolin and metronidazole
- Cefuroxime and metronidazole
- Ampicillin-sulbactam
- Clindamycin in those allergic to beta-lactam drugs.

Consequences of wound infection:

As a consequence of wound infection following surgery, there was an increase in the total duration of hospital stay, the total cost of treatment and the delay in initiation of post operative adjuvant radiation therapy.

The median duration of hospital stay in patients who had wound infection increased from 18 days to 34 days(19). Penel et al also studied the prognostic significance after head and neck cancer surgeries. In 95 patients studied the total wound infection rate was 50.5%, the median duration of post operative stay in patients was 15 days, which in the setting of wound infection went up to a median of 29 days. There was also a delay in the initiation of radiation in 21 out of 33 patients that required radiation therapy(55).

Inspite of the large amount of morbidity following these surgeries, the overall prognosis and outcome were the same irrespective of wound related complications.

TRICLOSAN:

Triclosan is a chlorinated, aromatic compound with the chemical name trichlorohydroxydiphenyl ether. The product was registered as a pesticide by the Environmental protection agency in 1969(56).

Triclosan was introduced as a antimicrobial agent into the industrial market in 1972(57) and its use was confined to the health care sector as a component in surgical scrubs(58). Over the coming years following approval from the Food and Drug Adminstration(FDA) and the Environmental Protection Agency(EPA) triclosan found broad application in the commercial market due to its antimicrobial properties. It thereafter has been incorporated into numerous personnel care and commercial products including soaps, toothpastes, cosmetics, deodorants, kitchen ware and children toys(56) as its action is efficacious against organisms in the oral cavity and the skin.

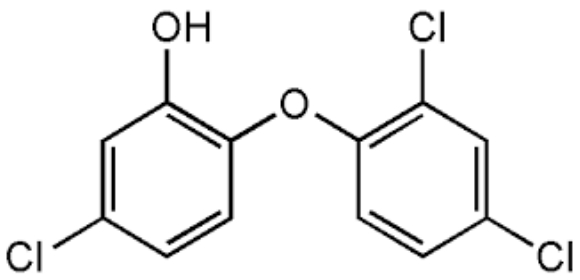
The organic compound in addition to its antibacterial properties, also exhibits antifungal action. At higher concentration, it acts as a bactericidal agent while it has bactertiostatic properities at lower concentrations.

Chemistry and Mechanism of action:

Triclosan is a chlorated bisphenol which is classified as a Class III drug by the FDA. The organic compound is a white solid powdered solid which has a mild phenolic odour. The compound contains two functional groups namely the phenol and the ether group.

The chemical formula : $C_{12}H_7Cl_3O_2$

Molecular structure:



Triclosan acts as an antagonist to the active site(FabI) of the enoyl-acyl carrier reductase enzyme. The enzyme is essential in the fatty acid synthesis in bacteria, which is crucial for cell wall synthesis and cellular functionality of the bacteria(59). The formation of this FabI - NAD^+ - Triclosan complex is responsible for the efficacy of triclosan as an antibacterial agent(60).

Safety and Use in humans:

The enoyl-acyl reductase enzyme is absent in humans and thus renders triclosan fairly safe for use in humans. The routes of administration to humans are either predominantly oral or dermal.

Metabolism of triclosan within the body:

When used orally either in the form of toothpaste or mouthwashes, there 0.08mcg/g of saliva of triclosan for upto 8 hours(61). In a study preformed using humans and rats after 24hours of local application of dermal triclosan, humans demonstrated 6.3% absorption as compared to 23% absorption seen in

rats. As the compound passes through skin, it is metabolized in the liver where it undergoes both glucuronidation and sulphation and is excreted via the renal system as triclosan glucuronide and triclosan sulphate. There were no active oxidative metabolites present in urine samples or in dermal samples(62). When administered orally and once absorbed, the plasma concentration are raised within 1-3hours. The terminal plasma half life is approximately 21 hours and within the first 24hours most of the triclosan has been eliminated. The baseline urinary concentration was reached within 1 week and 54% of the total triclosan was eliminated in urine within 4 days(63).

Triclosan showed high concentrations in the liver followed by the adipose tissues. The bioaccumulation for triclosan is low which confers safety in its usage.

Toxicity – There are rare reports of acute toxicity in terms of contact dermatitis and photosensitivity following exposure to triclosan(64).

Environmental Risks

Triclosan is water and lipid soluble and involatile. The cause for the high concentration of triclosan is secondary to high urinary concentration which enters the sewage system directly from unmonitored use of triclosan in commercial industries. During the water treatments, there is biodegradation of

triclosan of which a low level is desorbed from this aqueous form to become a surface water effluent. These effluents have a tendency to attach to any solid sediments within the aqueous medium resulting in bioaccumulation and poses a potential risk to aquatic life, invertebrates and certain fish(65),(56).

In waste water effluents, there are present chlorated derivatives of triclosan present either during the waterwaste disinfection with chorine or by skipping the standard treatments, these chlorinated triclosan derivatives can cause more endocrine dysfunction in addition to more antibacterial action. Triclosan derived dioxins are also formed after triclosan containing water has been treated with chlorine and then exposed to UV irradiation. These dioxins are more toxic than their parent compounds and can further breakdown to form highly chlorinated toxic dioxins, which are harmful to aquatic life and fish(66).

These environmental risks have contributed greatly in the past few years in raising bans against the indiscriminate use of triclosan.

Target organisms:

Triclosan has a broad spectrum of organisms that it is effective against including gram positive and gram negative non sporulating organisms. It is most effective against Staphylococci, some streptococci, few mycobacterium, enterococci and proteus species. It also has some fungicidal activity. Any

bacteria or micro-organism containing the FabI site on the enoyl-acyl reductase enzyme is susceptible to triclosan(57). Triclosan thus acts against organisms that colonize the skin and the oral cavity which when impregnated into medical devices and sutures may contribute to decreasing the risk of surgical site infections. Sutures, urinary catheters, central venous catheters and orthopaedic implants when impregnated with triclosan have been used and researched in the medical industry since the 1980s.

Different microorganisms affected by the antimicrobial action of TCS(66).

Target Microorganisms	**Effective Concentrations**
Most sensitive strains	
Staphylococci, some Streptococci, some mycobacteria, *Escherichia coli*, *Klebsiella pneumonia*, *Klebsiella* spp., *Enterobacter* spp., *Acinetobacter* spp., *Proteus* spp. and *Proteus mirabilis*, *Plasmodium falciparum*, *Toxoplasma gondii*	0.01 $mg \cdot L^{-1}$ to 0.1 $mg \cdot L^{-1}$
Less sensitive strains	
Methicillin-resistant *Staphylococcus aureus* (MRSA) strains	0.1–2 $mg \cdot L^{-1}$
Enterococci	-

Target Microorganisms	Effective Concentrations
Highly resistant strains	
Pseudomonas aeruginosa, Clostridium difficile	-

Triclosan coated sutures:

For over 30 years, the question whether sutures act as a nidus of wound contamination and infection has been debatable. As with the use of any other biomedical implant, sutures too can cause tissue inflammation, microbial adherence and bacterial colonization. Bacterial affinity varies with the type of suture material used. Studies have shown that bacteria are more adherent to braided sutures like silk, polyglactin when compared to monofilament sutures like nylon. The bacterial organisms studied included *Staphylococcus aureus, Staphylococcus epidermidis, Methicillin resistant staphylococcus aureus* and *Escherichia coli*(67,68). Tissue reactions occur when biomedical devices and suture material are introduced into the body. The surface of the suture gets coated by proteins as part of the tissue reaction, these proteins – fibronectin, fibrinogen and collagen which act as adhesions for bacteria(68). Bacteria that colonize the skin surface may be introduced into the wound tracks, where skin colonizes like staph. Epidermidis can form a biofilm which confers protection upto the organism from the body's defense mechanisms. Triclosan is effective against the bacterial colonization and thus the formation of the biofilm.

The incorporation of triclosan into suture was found to perform the same if not better than the uncoated polyglactin. The intra-operative handling, the ease of throwing a knot, the holding of the first knot, the memory and its passage through tissues were studied and found to be the same as the plain uncoated polyglactin sutures(69)(70). There was a definitive difference in the immediate post operative pain in those where the coated sutures were utilized. Pain in the immediate post operative period is an indication of early sub-clinical infection, whether the decrease in the pain was due to inhibition of the colonization of the bacteria is left to speculation(69).

Antibiotic Resistance:

There have been many theories regarding the development of microbial resistance to triclosan. These include(66):

- Overproduction of targets
- Modification of the targets
- Membrane permeability and barriers
- Efflux pump(71)- as seen in Pseudomonas aeruginosa.
- Over-expression of the target enzyme
- Non susceptibility of the target enzyme or alteration to the FabI enzyme.

Pseudomonas is unique in that it possess both sensitive and resistant enzymes – FabI, FabK. It also has intrinsic resistance due to expression of multiple efflux systems. Only four such systems have been characterized of which the MexAB-OprM efflux system is responsible for triclosan resistance(71). E.Coli when exposed to triclosan is capable of selection of FabI mutants and causes overproduction of FabI which leads to an increase in the triclosan resistance. Strains of staphylococcus aureus which showed decreased sensitivity to triclosan were found to have a mutation in the FabI enzyme which resulted in overproduction of these strains(72)(73).

Triclosan is a substrate of several MDR efflux pumps and may promote multidrug resistance in bacteria to both the antiseptic and antibiotics, thus compound antibiotic resistance. The fear of overuse of triclosan is cross-resistance which will promote the emergence of super-bugs.

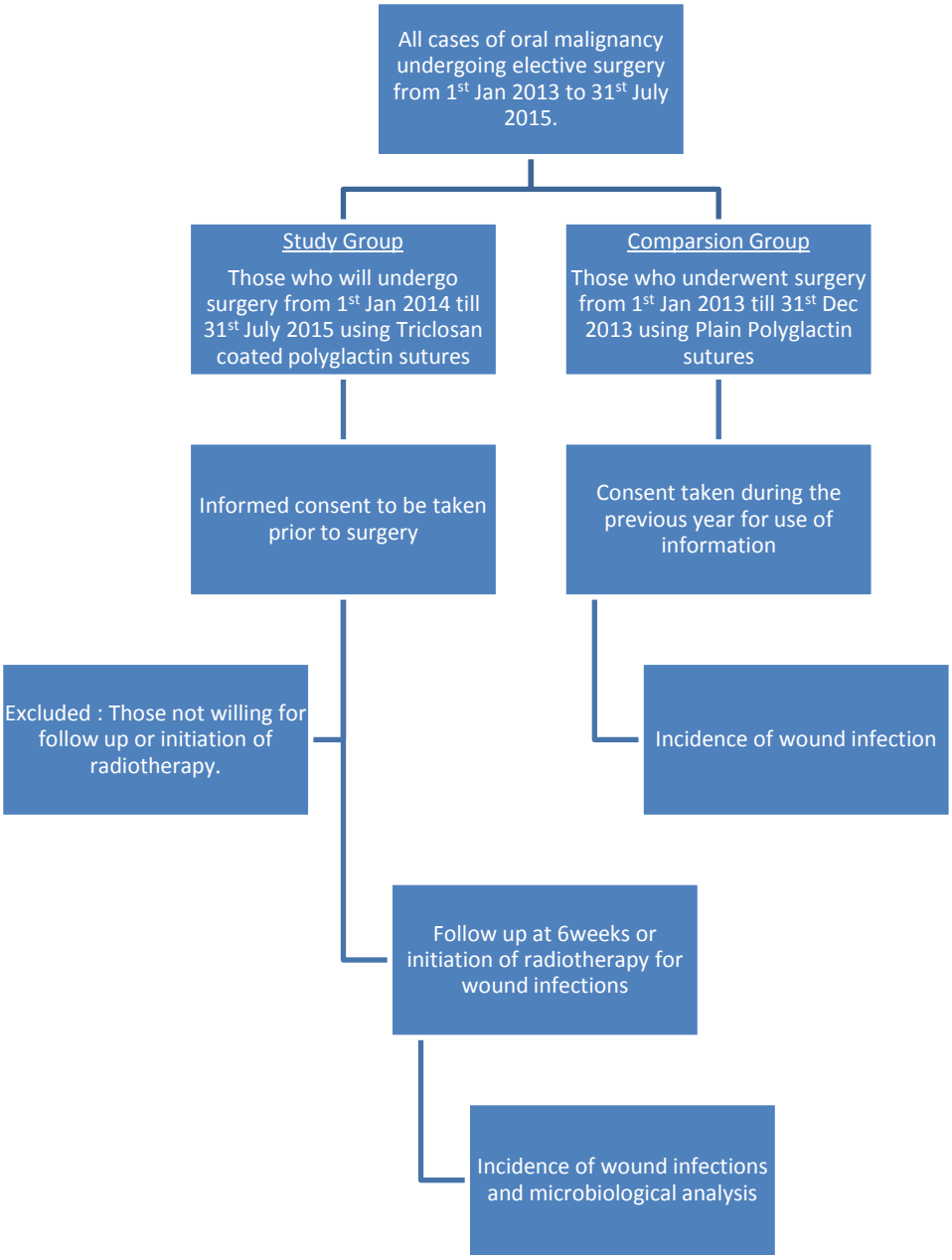
Drug resistance in the laboratory and in the environment was different. Studies revealed that use of triclosan in hygiene products did not affect the nature of the oral flora or change their susceptibility to antibiotics. There has been no established relationship between triclosan usage in practice and development of antibiotic resistance(74). The use of triclosan should be restricted and closely monitored as there exist a potential for resistance in the future.

Triclosan has no known mutagenic, carcinogenic or any associated toxicities. As the molecular structure closely resembles that of the thyroid and oestrogen molecules, there has been concern regarding disruption of the endocrine and reproductive axis. The potential hazards and risks are currently still currently being investigated by the EPA and the FDA.

METHODS

A pilot study was done to determine the incidence of wound infection in consecutive cases that undergo surgery for oral malignancies with all surgical sites being closed with triclosan coated polyglactin 910 sutures from 1st January 2014 to 31st July 2015.

Diagrammatic Algorithm of the study



Methods in detail:

i. **Intervention and Comparator agent –** Antimicrobial coated polyglactin sutures versus plain polyglactin sutures

ii. **Key Criteria**

Inclusion Criteria:

- All patients undergoing elective surgery for oral malignancy.
- Patients willing for follow up at CMC, Vellore
- Patients willing to be followed up till initiation of radiotherapy.

Exclusion Criteria:

- Patients not consenting.
- Patients not willing for follow up in our institution.
- Patients not willing for radiotherapy at our institution.

iii. **Method of randomization:**

- All consecutive cases over a period of one and a half years, starting from 1st January 2014 till 31st July 2015.

iv. Method of allocation concealment: nil

v. Blinding and masking: nil

vi. Primary Outcome:

- To determine the incidence of wound infection in both study groups

vii. Secondary Outcome/s:

- To assess the duration of hospital stay.
- To find the delay in initiation of radiotherapy in those with wound infection.
- To do a microbiological analysis of the oral cavity in oral malignancy cases.
- To evaluate the bacteriology of wound infections involved when operating these patients.

viii. Target sample size and rationale:

The sample size was calculated using the software nMaster 2.0

The proportion in group A is 0.38(75)

The expected proportion in Group B is 0.20

Risk Difference = 0.18

Alpha error = 5%

Power = 80%

The minimal sample size required to compare the infection rates in both groups is = 90

So total number = 90 + 90 = 180

So total sample size required for the study is 180.

The formula used for sample size calculation is:

Formula

$H_o : P_1 = P_2$; $H_a : P_1 \neq P_2$

$$n = \frac{\left\{ Z_{1-\frac{\alpha}{2}} \sqrt{2\,\bar{P}\,(1-\bar{P})} + Z_{1-\beta} \sqrt{P_1\,(1-P_1) + P_2\,(1-P_2)} \right\}^2}{(P_1 - P_2)^2}$$

Where,

$$\bar{P} = \frac{P_1 + P_2}{2}$$

P_1 : Proportion in the first group

P_2 : Proportion in the second group

α : Significance level

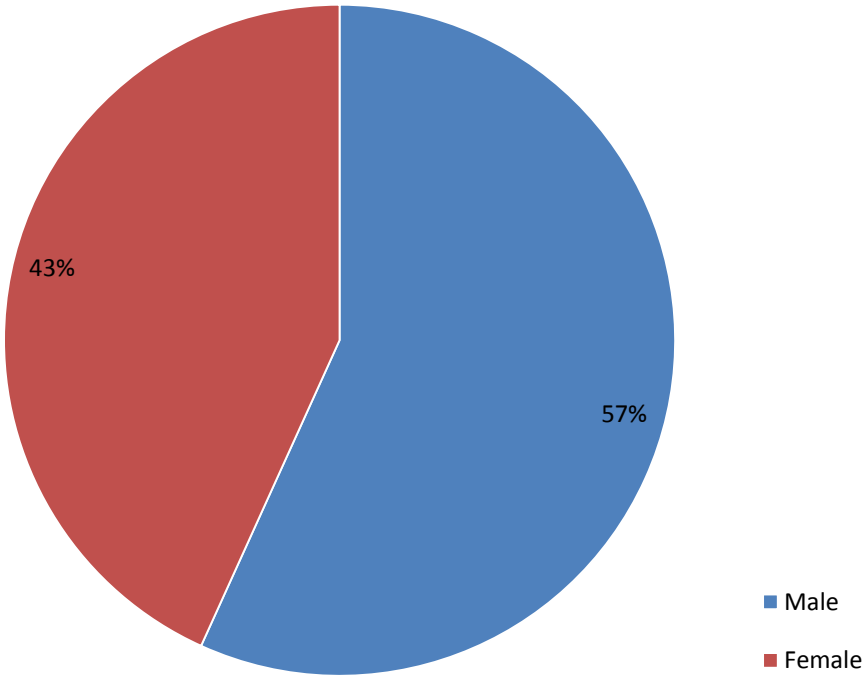
$1-\beta$: Power

RESULTS:

A total of 53 patients were recruited for the study. The patients for the study were all recruited prior to surgery from the surgical wards and the outpatient department.

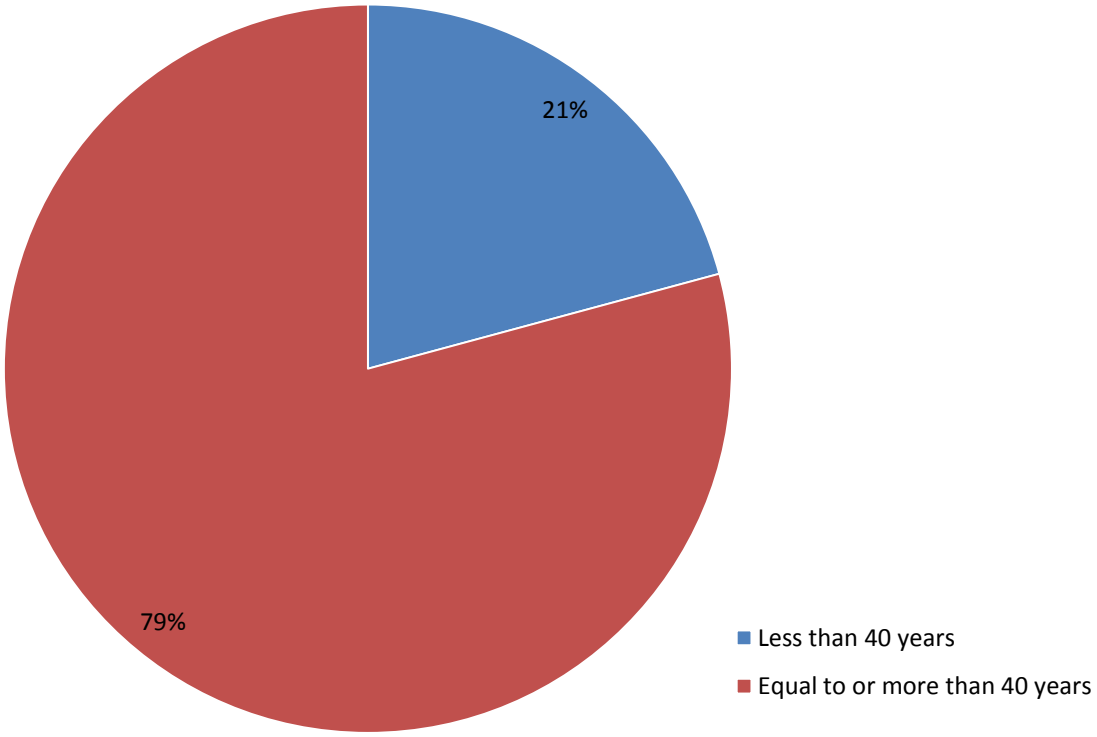
PROFILE OF PATIENTS:

Fig 1: Sex Distribution



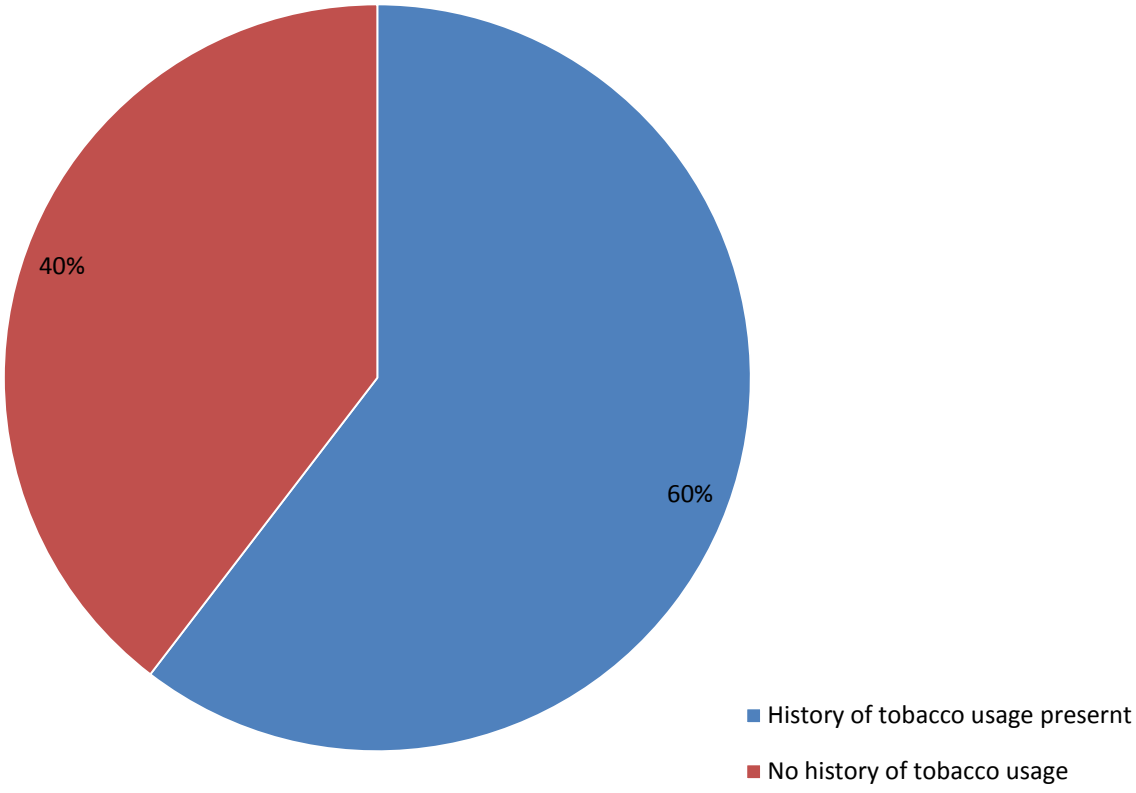
The total cases recruited were 53, of which 31 (57%) were males. The total number of females included in the study was 22 (43%) as seen in Fig 1.

Fig 2: Age distribution



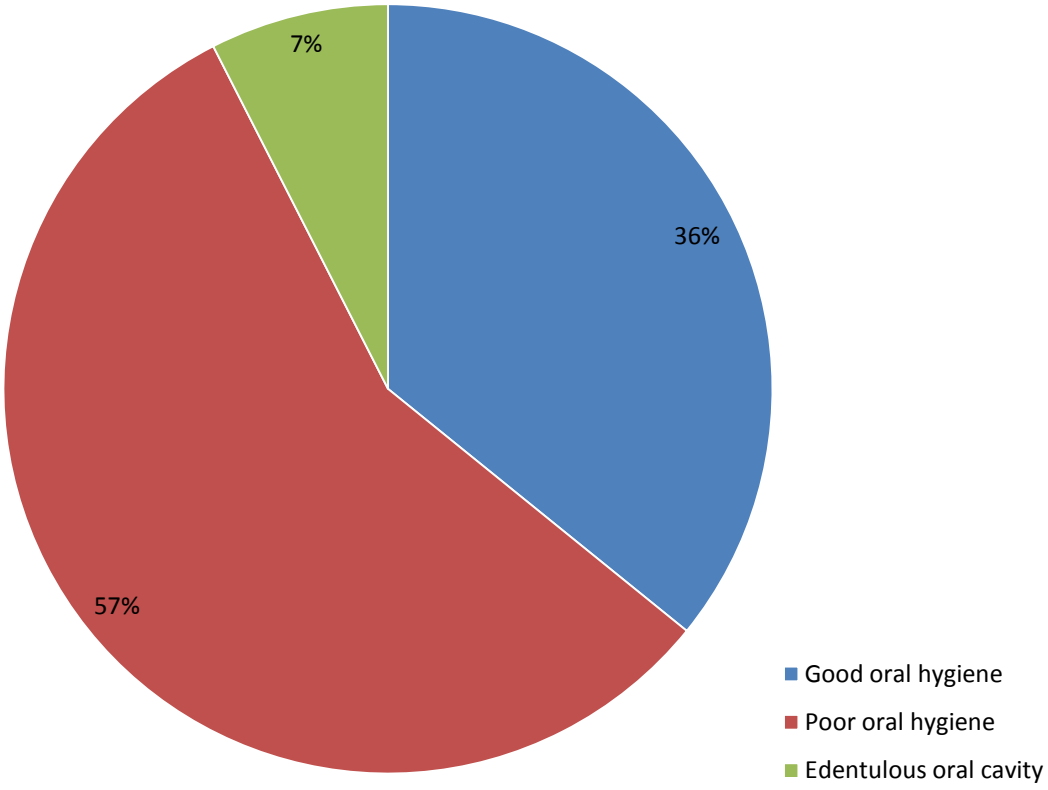
There were 42 patients equal to or above the age of 40 years(79%) which constituted the majority. The number of those below the age of 40 years was 11 (21%).

Fig 3: Tobacco usage



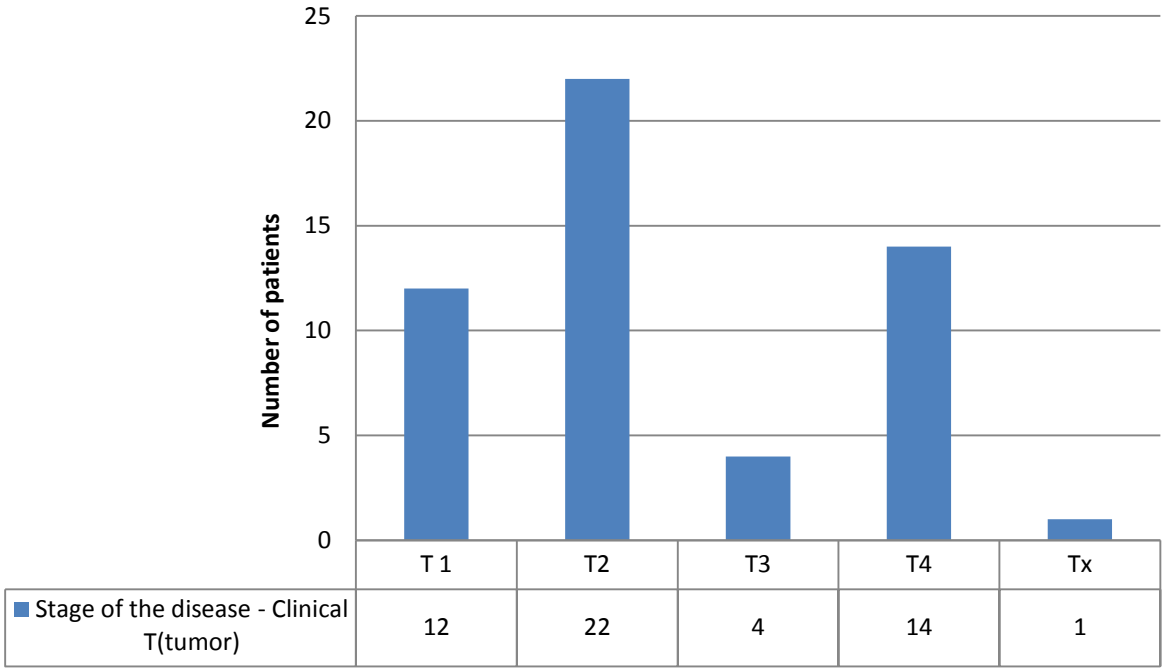
Of all the patients recruited 60% of patients gave a positive history of tobacco usage either in the form of cigarette smoking, chewing of paan with tobacco or using a tobacco quid.

Fig 4: Dental status



Of the total of 53 patients recruited, only 36% (19 patients) have good oral hygiene as documented by no oral cavities, plaques or staining of teeth. Of the 53 patients, 57% of patients, 30 patients had bad or poor oral hygiene – presence of plaques, staining or dental caries. There were 4 patients of the total 53 were edentulous. Whether or not the dental status was a cause for an increased infection rate was studied by doing a univariate analysis.

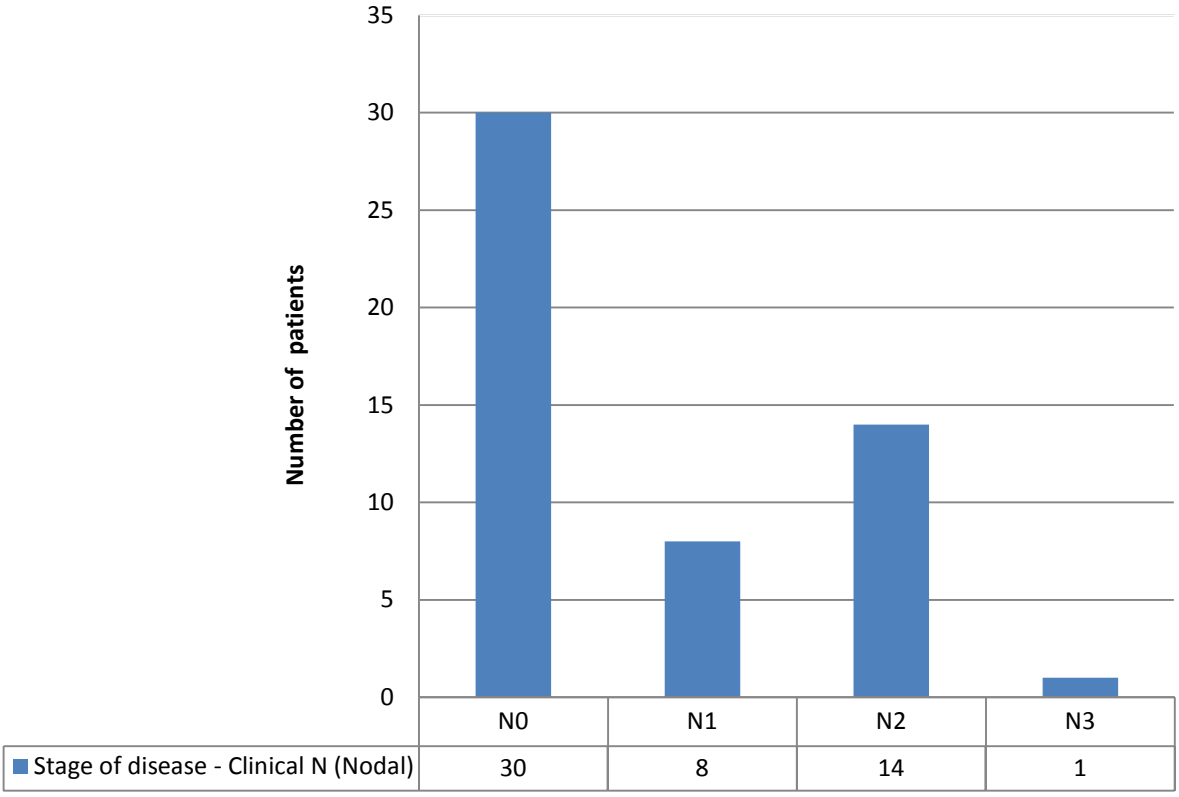
Fig 5: Stage of the disease - Clinical T(tumor)



	T 1	T2	T3	T4	Tx
Stage of the disease - Clinical T(tumor)	12	22	4	14	1

The oral cavity disease was assessed clinically and was staged according to the AJCC, TNM staging for oral malignancy. All the 53 patients, there were 41.5% diagnosed to have T2 and 26.4% were diagnosed to have T4 disease.

Fig 6: Stage of disease - Clinical N (Nodal)



Following clinical evaluation and staging by the AJCC TNM staging, 30 (56.6%) patients had N0 disease. N1 disease was seen in 15.1% of total cases and N2 and N3 disease accounted for 26.4% and 1.9% of all cases respectively.(Figure 6). The relationship of the clinical nodal disease was compared with the infection rate in the univaritate analysis.

Fig 7: Stage of disease - Pathological T (tumour)

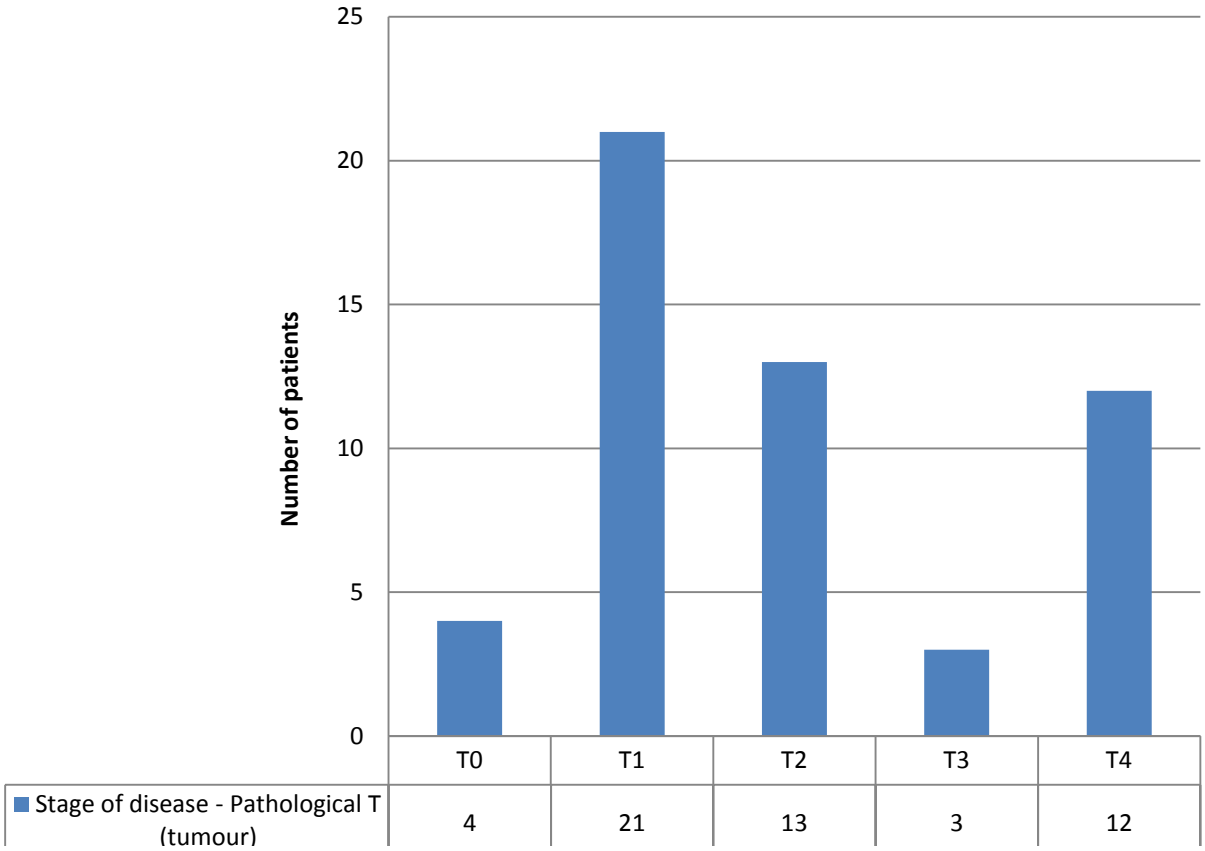


Figure 7 shows the frequency distribution of the patients in terms of the pathological T stage of the tumour. T1 cancers constituted 39.6%, T2 cancers accounted for 24.5% of all cases while T4 cancers accounted for a total of 22.6%.

Fig 8: Stage of disease - Pathological N (nodal)

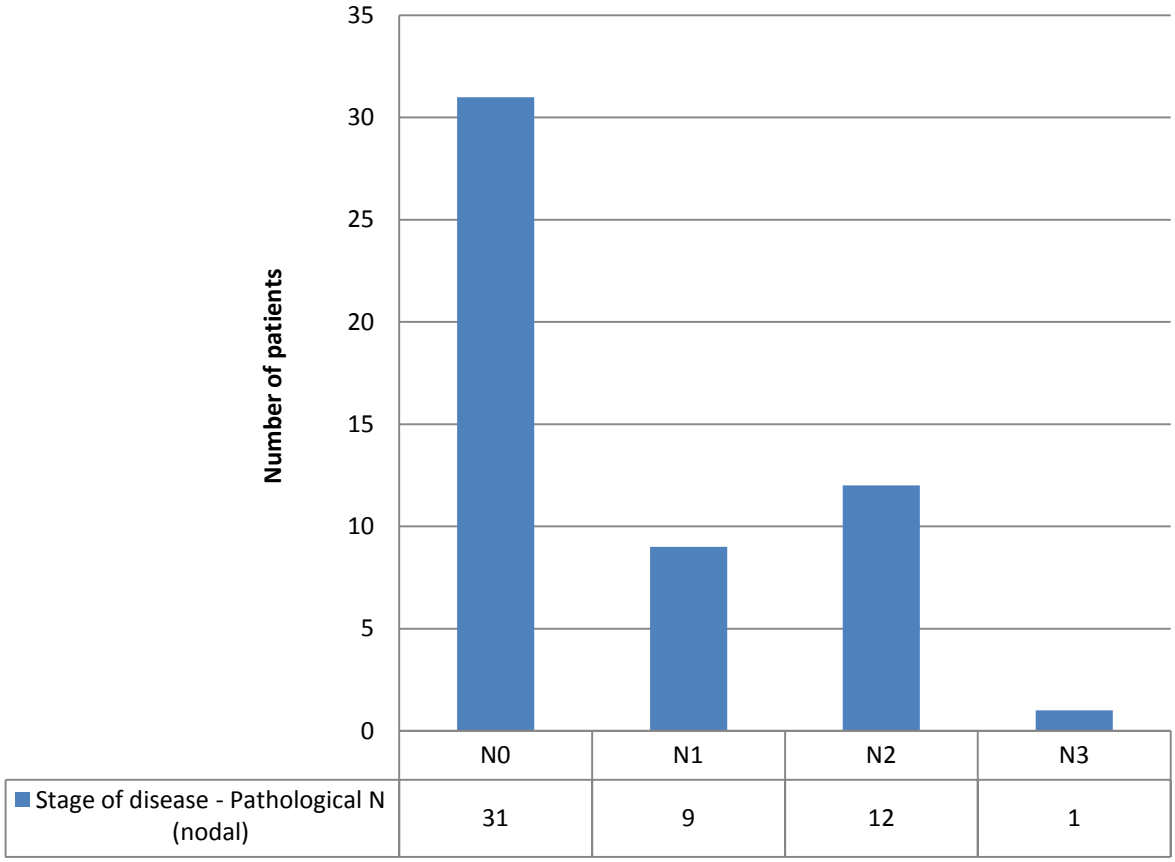
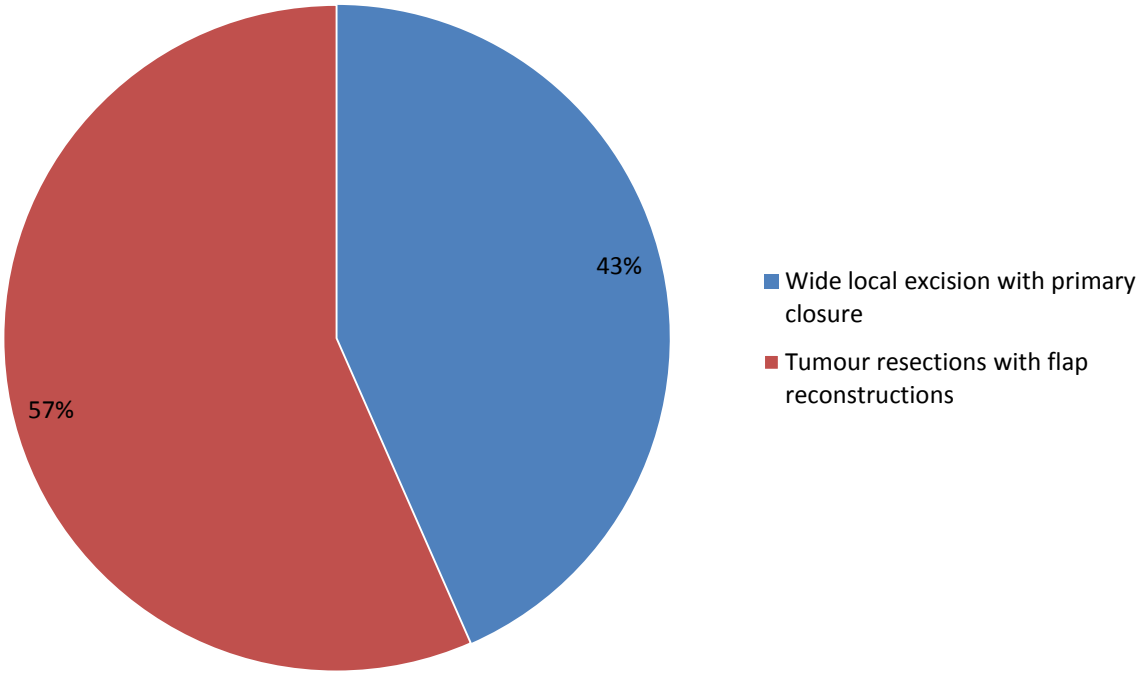


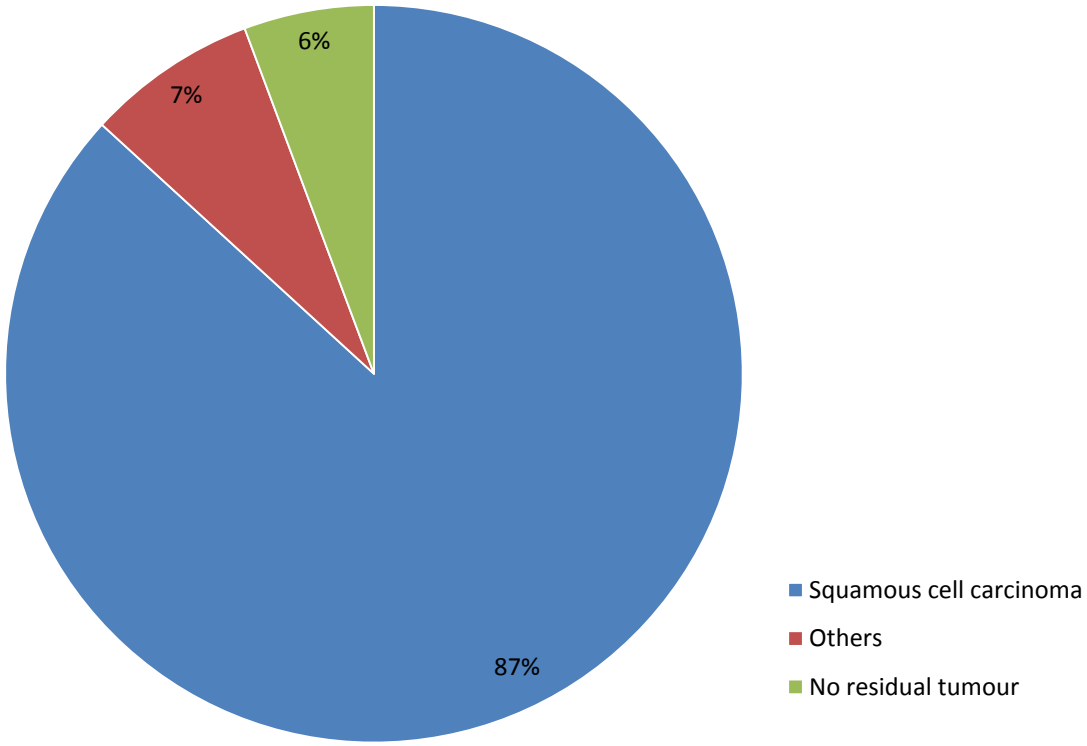
Figure 8 shows the frequency distribution of the pathological N stage of the tumour. N0 cancers were seen in 54.7%,andN2 cancers accounted for 22.6% of all cases while N1 cancers accounted for a total of 17.0%.

Fig 9: Operative procedure preformed



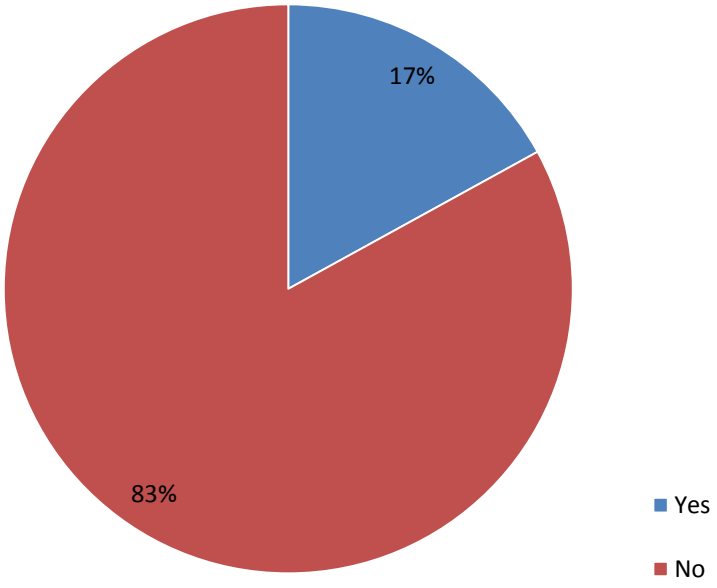
As seen in the Figure 9, the distribution of cases was recruited based on the type of surgery performed. Early lesions were treated with a wide local excision and primary closure. More advanced lesions required tumour resection with flap reconstructions and either unilateral or bilateral neck dissections. The flap reconstructions included both myocutaneous flap – pectoralis major, delto-pectoral flap, – bi-paddle flap and microvascular free flaps – radial forearm free flap, fibula free flap.

Fig 10: Histopathology of the resected tumour



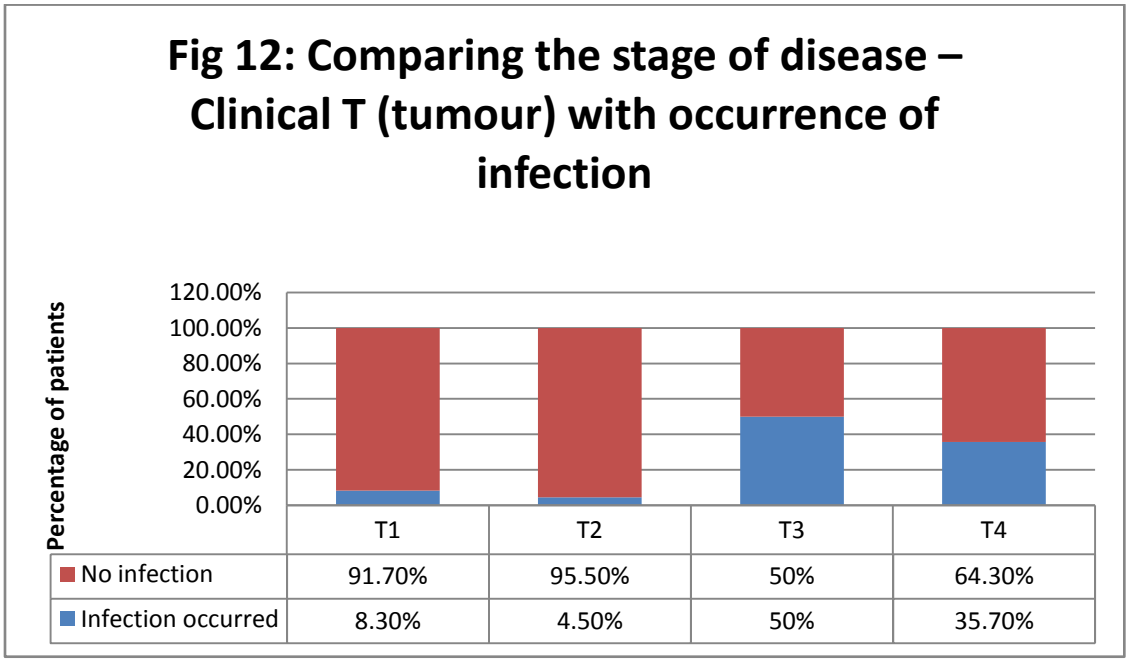
The majority of the oral cancers were squamous cell carcinoma as was consistent with other studies. Of all the patients recruited, 3 patients (6%) did not have any residual tumour either following preoperative chemoradiation or from previous wide local excisions with positive margins. 4 patients had non squamous pathology which included ameloblastoma, adenoid cystic carcinoma, verrucous carcinoma and rhabdomyosarcoma.

Fig 11: Occurrence of post operative infection while using triclosan coated sutures



Triclosan coated polyglactin 3-0 suture on a cutting needle was used either in continuous or interrupted sutures to close all the intraoral suture lines. There was no change in the technique of the operative procedure when compared to the previous year. There was no difference in suture handling as reported by the operating surgeons when compared to uncoated polyglactin sutures. A total number of 53 patients were recruited for the study and a total of 9 patients developed a surgical site infection as per the definition by the Center for Disease Control.

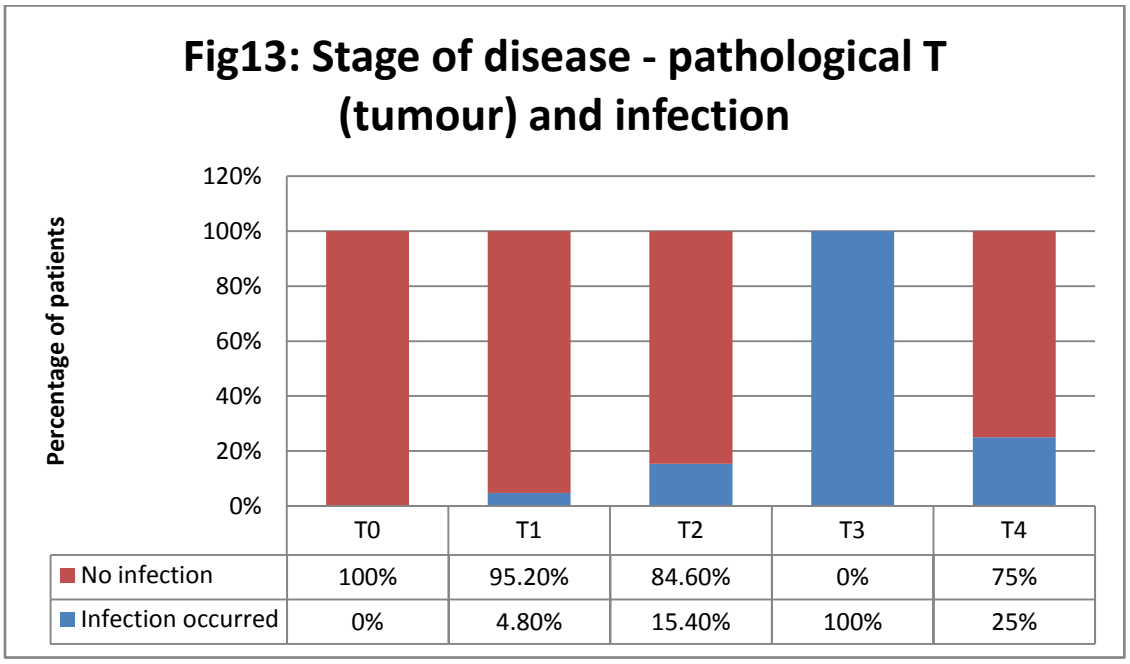
Univariate analysis:



Stage of disease – clinical T (tumour)	Infection occurred (no of patients)	No infection (no of patients)
T1	1	11
T2	1	21
T3	2	2
T4	5	9

Table 1: Comparing the stage of the disease – clinical T stage with number of patients with infection

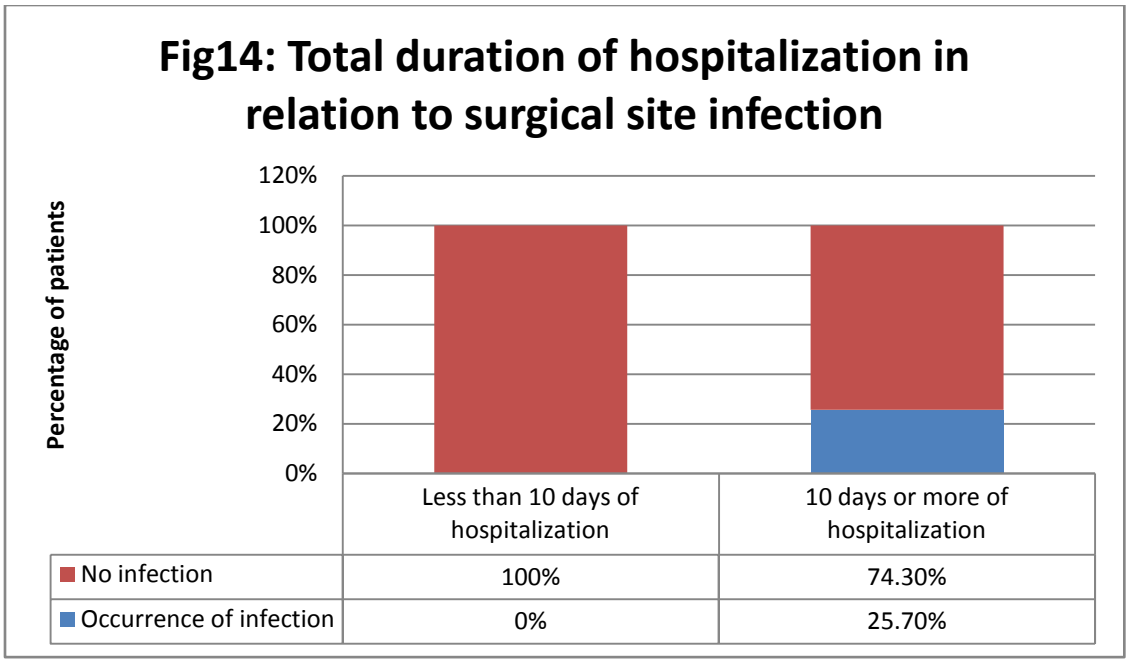
As seen in Table 1, T4 tumours had the highest number of total patients with wound infection.The incidence of post-operative wound infection attained statistical significance as the clinical stage of the tumour increased with P = 0.024 ($p<0.05$, confidence interval = 95%).



Stage of disease – pathological T (tumour)	Infection occurred (no of patients)	No infection (no of patients)
T0	0	4
T1	1	20
T2	2	11
T3	3	0
T4	3	9

Table 2: Stage of the disease – pathological T with relation to the number of patients with wound infection

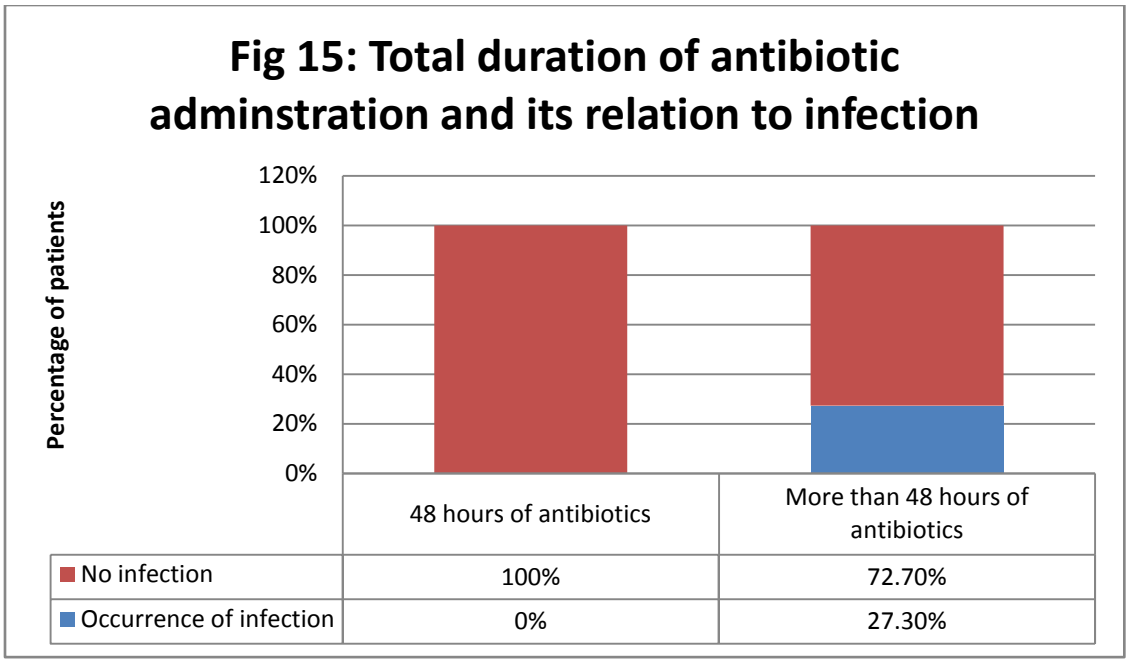
Figure 2 shows the correlation of the pathological T-stage and the incidence of wound infection in the study population. These findings are in consistency with the causal relationship between the pathological T stage of the disease and the incidence of wound infection with p = 0.001 (confidence interval of 95%).



Total duration of hospital stay (days)	Infection occurred (no of patients)	No infection (no of patients)
Less than 10 days	0	18
10 days or more	9	26

Table 3: Relationship between the total duration of hospitalization in relation to number of patients with surgical site infections

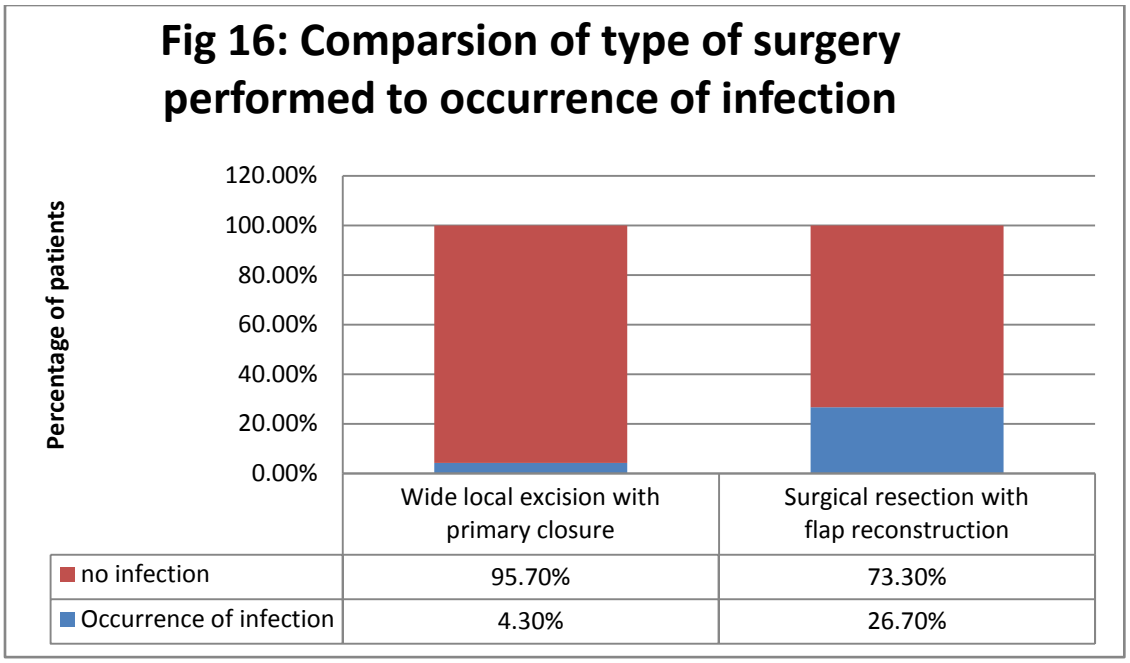
The total duration of hospitalization is increased in those patients who have wound infections, this was statistically significant, $p = 0.01$ ($p<0.05$, confidence interval of 95%). The conclusion is that in those who have a wound infection required a longer duration of hospitalization and in effect an increase in the cost of treatment. The Table 3 shows that all the 9 nine patients who had post operative infections were admitted for a duration of more than 10 days.



Total duration of antibiotics	Infection occurred (no of patients)	No infection (no of patients)
48 hours of antibiotics	0	20
More than 48 hours of antibiotics	9	24

Table 4: Shows that all the patients with wound infections had received antibiotics

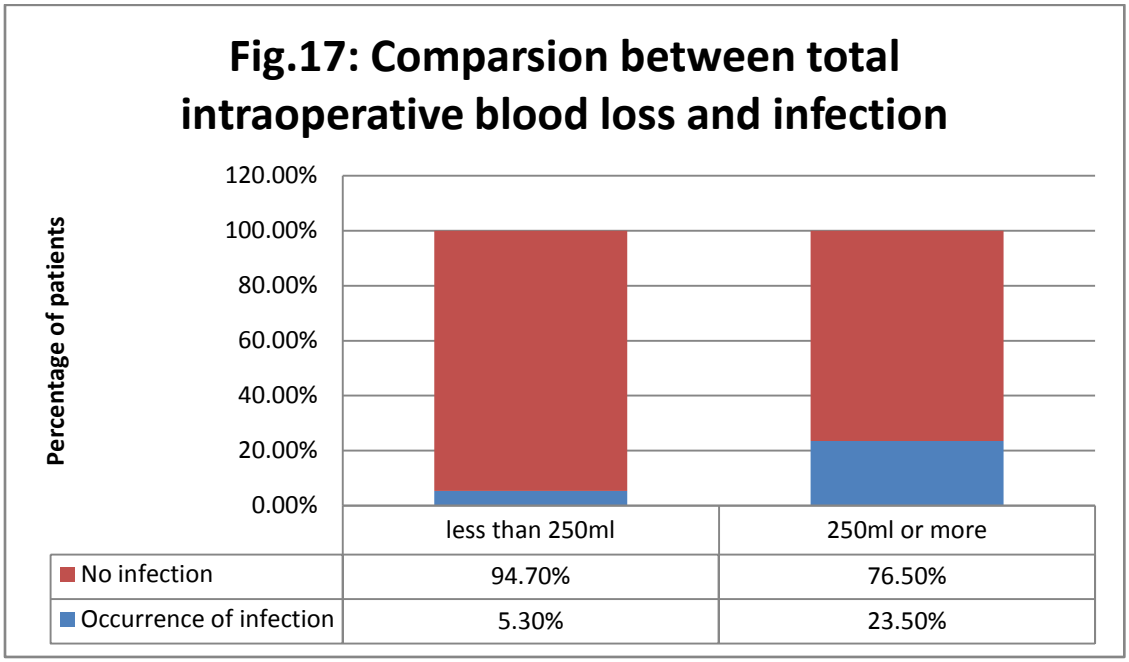
There was an association with total duration of antibiotics and incidence of wound infections. The patients who received 48 hours of antibiotics also underwent less extensive surgeries, when compared to those who received more than 48 hours of antibiotics. for more than 48 hours, this was statistically significant with $p = 0.01$ ($p<0.05$, confidence interval of 95%).



Type of surgery preformed	Infection occurred (no of patients)	No infection (no of patients)
Wide local excision with primary closure	1	22
Surgical resections with flap reconstruction	8	22

Table 5: Type of surgery performed in comparison to number of patients with infection

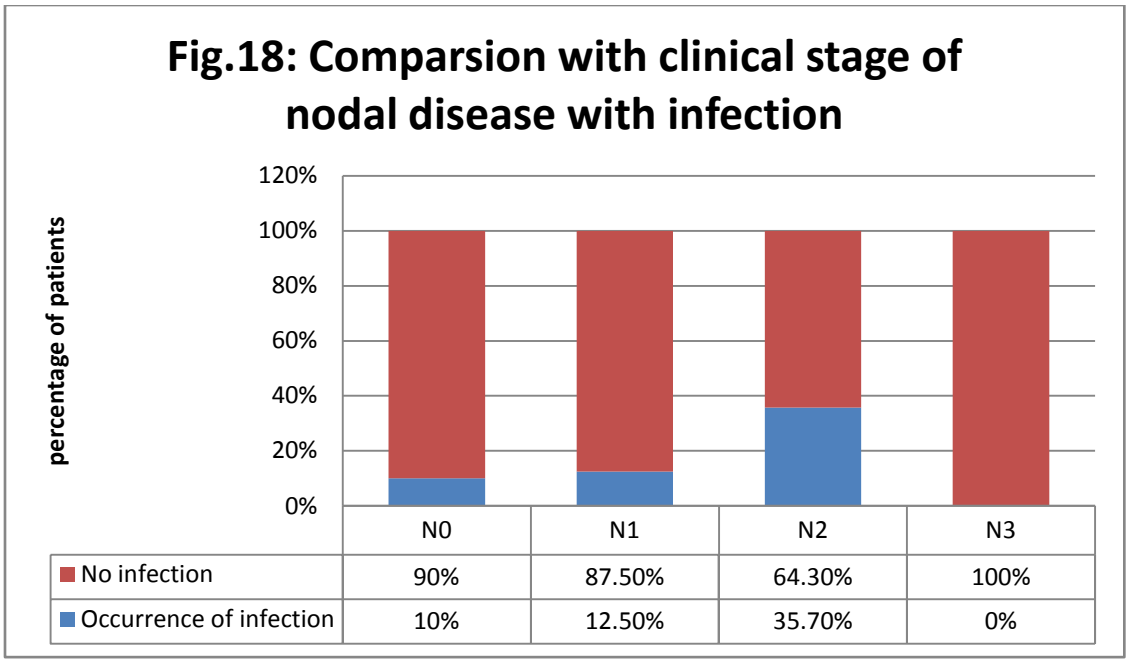
As the extent of surgical resection increases there is an associated increase in the infection rate, $p = 0.061$ ($p < 0.1$, confidence interval of 90%). Those who underwent surgical resections with flap reconstructive surgeries are at a higher risk of wound infections. This includes reconstructions done with both myocutaneous flaps and micro-vascular free flaps. The inference to check for statistical significance at 5 % can occur only once the total sample size is complete.



Intraoperative blood loss	Infection occurred (no of patients)	No infection (no of patients)
Less than 250ml	1	18
250ml or more	8	26

Table 6: Comparison of intra-operative blood loss and its correlation to number of patients with infection.

There was an increase in the number of patients with infection when there was more than 250ml of intra-operative blood loss, which was statistically significant at an alpha error of 20% (p = 0.133) . Whether this is statistically significant at an alpha error of 5% can be determined only on completion of the proposed sample size.

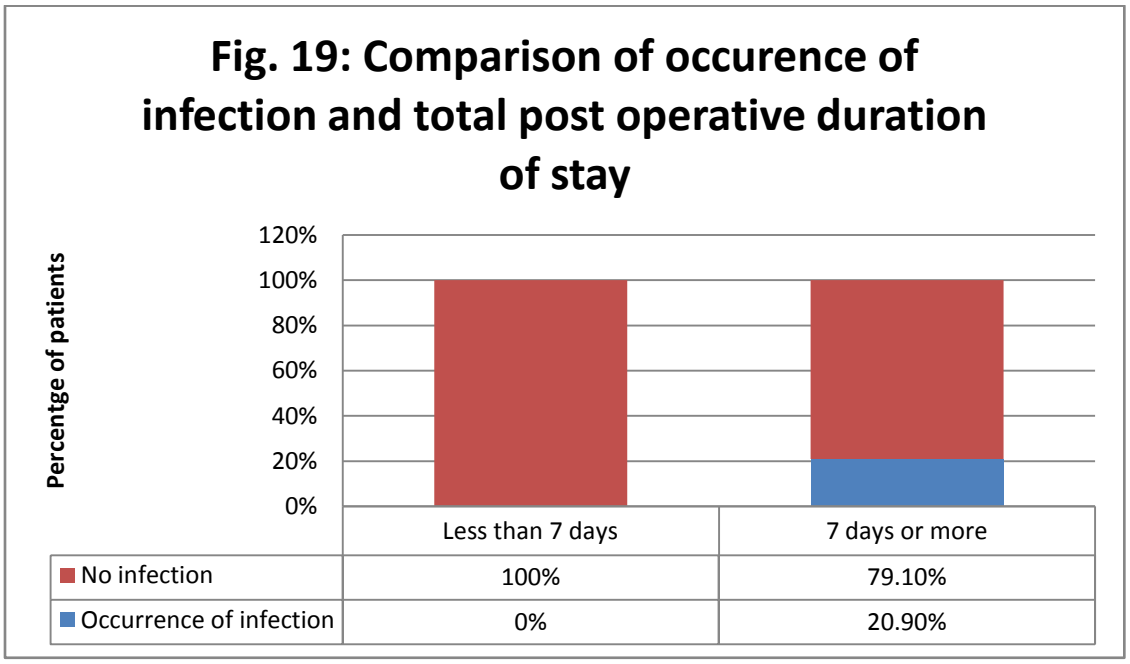


Stage of disease – clinical N (nodal)	Occurrence of infection (no. of patients)	No infection (no. of patients)
N0	3	27
N1	1	7
N2	5	9
N3	0	1

Table 7: Comparison of clinical stage of nodal disease with number of students with infections

There was an association with an increase in wound infection rate as the clinical nodal stage increased (Table 7), though there was no statistical significance with $p = 0.184$. If allowable alpha error was taken as 20%, with a confidence interval of 80%, then $p = 0.2$, then clinical nodal disease would be statistically significant. As nodal disease increases so does the extent of surgery performed with more extensive nodal dissections. As the current sample size is inadequate

for this assessment, to fully understand the significance, it would warrant completion of the proposed study.



Total duration of post operative stay	Occurrence of infection (no. of patients)	No infection (no. of patients)
Less than 7 days	0	10
7 days or more	9	34

Table 8: Comparison of infection rates with total duration of post operative stay

There is an association between the occurrence of infection and an increase in the duration of post operative stay. The p value calculated from the above Table 8 is 0.18, which on allowance of an alpha error of 20%, and a confidence interval of 80% is statistically significant. Significance at a confidence interval

of 95% will require the completion of the proposed pilot study, as the sample size currently achieved is inadequate for this assessment.

Multivariate analysis:

On doing a multivariate analysis, there was no significant risk factor that was associated with an increased infection rate. To get any conclusive results the sample size will have to be completed following which analysis will have to be done.

There were multiple other factors that were studied but were found to be statistically insignificant.

	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Male Sex	4 /12.9%	27 / 87.1%
Female Sex	5 / 22.7%	17 / 77.3%

Table 9: Comparison of sex distribution with incidence of infection

Here in Table 9, the calculated p = 0.464 which was statistically insignificant.

	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Less than 40 years	1 / 9.1%	10 / 90.9%
40 years or more	8 / 19.0%	34 / 81.0%

Table 10: Comparison of age of patients with incidence of infection

The p value calculated from the Table 10was 0.665, which was not significant.

Thus age had no bearing to incidence of wound infections.

	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Diabetic	1 / 20.0%	4 / 80.0%
Non - diabetic	8 / 16.7%	40 / 83.3%

Table 11: Comparison of history of diabetes and infection rates

Here the calculated p = 1.000, which was more than 0.05 and was thus

insignificant.(Table 11)

	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Tobacco usage	4 /12.5%	28 / 87.5%
No tobacco usage	5 / 23.8%	16 / 76.2%

Table 12: Comparison of tobacco usage and its link to occurrence of infection

The p value derived from the Table 12 was 0.456, these findings were statistically insignificant, and thus there was no relationship between tobacco use and occurrence of infection.

History of chemotherapy/ Radiation	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Yes	0 / 0%	3 / 100%
No	9 / 18.0%	41 / 82.0%

Table 13: Comparison of previous radiation therapy or chemotherapy with incidence of wound infection

There was no increase in infection if there was history of previous chemotherapy or radiation. In the Table 12, the calculated p = 1.000 which was insignificant statistically.

Hemoglobin (mg/dl)	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Less than 8	0 / 0%	1 / 100%
8 or more	9 / 17.3%	43 / 82.7%

Table 14: Comparison of hemoglobin preoperatively of the patient with incidence of wound infection

In Table 14, the calculated p = 1.000, this was less than 0.05 and was insignificant. The relationship between the pre-operative hemoglobin levels to occurrence of infection was absent.

Total platelets	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Less than 4 lakh	3 / 10.7%	25 / 89.3%
4 lakh or more	1 / 50.0%	1 / 50.0%

Table 15: Comparison of total platelets preoperatively with incidence of wound infection

In Table 15, the calculated p = 0.253, which is statistically insignificant. There was thus no relationship between the total per-operative platelets and occurrence of infection.

Initiation of radiation therapy	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Less than 6 weeks	3 / 23.1%	10 / 7.9%
6 weeks or more	4 / 44.4%	5 / 55.5%

Table 16: comparison of initiation of radiation therapy and infection

In Table 16, the calculated p = 0.376, which is insignificant statistically.

Histopathology of tumour	Occurrence of infection (frequency/percentage)	No infection (frequency/percentage)
Squamous cell carcinoma	9 / 19.6%	37 / 80.4%
Others	0 / 0%	4 / 100%

Table 17: Incidence of wound infection with difference in the histopathology of the tumour

In the above Table 17, the calculated p = 1.000, and reveals no relationship between the incidence of wound infection and histo-pathological report of the tumour in itself.

MICROBIOLOGICAL ANALYSIS:

Oral swabs were taken preoperatively prior to administration of antibiotics in the operation theatre. In those individuals who developed wound infections, cultures were taken from the site of the infection and sent for analysis.

SL.NO	PRE-OPERATIVE	POST – OPERATIVE
1.	Staphylococcus aureus	Enterobacter, staphylococcus aureus
2.	Pseudomonas aeruginosa	Pseudomonas aeruginosa
3.	Enterobacter	Alpha hemolytic streptococcus
4.	Pseudomonas aeruginosa, Klebsiella	Klebsiella, Enterobacter
5.	Escherichia coli, streptococcus	Staphylococcus, Streptococcus
6.	Normal oral flora	Staphylococcus
7.	Beta hemolytic streptococcus	Staphylococcus aureus, Escherichia coli
8.	Non-hemolytic streptococcus, staphylococcus aureus, Fusobacterium	Streptococcus
9.	Klebsiella	Escherichia coli, non fermenting gram negative bacilli

In additional to the above organisms, there were also cultures that grew normal oral flora in the midst of oral malignancy. Whether more extensive oral lesions have a more varied oral flora is left to speculations and would require further, more extensive in detail evaluation.

DISCUSSION:

In our pilot study, 53 patients were recruited of which only 9 patients developed wound infections post operatively. This accounted for 17% during the course of the study. Prior to this, during the period of January 2013 to December 2013, a total of 88 operations for oral malignancies were done in our department, of which 21 (23.86%) patients had post operative wound infection. This was found to be statistically insignificant with a p=0.33 at the current sample size of 53 patients. The true statistical significance is awaited completion of the proposed sample size.

Triclosan coated sutures have found applications during the closure of the subcutaneous layer in major abdominal surgeries. Multiple studies have been done with varying conflicting results. In a case control study performed by Rasic Z et al, 184 patients were recruited with colorectal cancers, for 91 patients, triclosan coated polyglactin sutures were utilized. There were 12 patients who developed surgical site infections, the use of the coated sutures was shown to decrease wound infection in addition to total post operative duration of stay(76). Another multicenter randomized PROUD trial published in The Lancet in July 2014, found that incorporating triclosan into polydioxanone sutures did not reduce the incidence of surgical site infections(77). Wang et al conducted a systemic review and meta-analysis on 17 randomised control trials were 3720 patients were recruited. He found that

there was a 30% reduction in the total surgical site infections with the use of triclosan coated sutures(78). A meta-analysis was done to assess the prevention of infections following incision closure with Triclosan coated sutures. Daoud et al carried out the meta-analysis which included 15 randomized control trials with 4,800 study subjects. The results of the analysis suggest strongly that the presence of triclosan within the incision site was an important factor responsible for a decrease in the incidence of surgical site infections(79). It has also been clearly stated that in view of inadequate number of trial that include the operation type and the definition of a surgical site infection, it is prudent to continue these studies after considering these factors(79).

The cause for infections is multifactorial and there appears no one answer to why infections occur. These factors may either be patient related or other extrinsic factors. In our study, there was an increase in wound infection rate as the clinical T stage of the tumour increased, with 5 patients with T4 disease having wound infection, where $p = 0.001$ ($p<0.5$, CI 95%). This was statistically significant and consistent with findings by Penel et al(19) and Robbins et al(45). For all oral malignancies, patients have been advised to take prophylactic antibiotics. In our unit, for all early stage lesions, Metronidazole and Salbactum – Cerfeperazone were given for a duration of 48 hours. For Stage III and IV disease, the antibiotics were continued for 5 days. In our study, we found that those who received antibiotics for more than 48 hours had an

increased wound infection rate, p = 0.01 (p<0.5, CI 95%) as was reported by Lofti et al(75). These patients are also those who had higher staged tumours and more extensive surgical dissections.

An increase in the total duration of hospital stay was seen in patients who had wound infections, all nine patients who had a surgical site infection had a total duration of hospital stay of more than 10 days, p = 0.01 (p<0.5, CI 95%). The increases in duration of hospital stay also lead to an increase in the total hospital expenses which was not quantified in this study.

In patients who underwent surgical resections with flap reconstructions, there was an increase in the surgical site infection, p = 0.061 (p<0.1, CI 90%), similar findings were reported by Belusic-Gobic et al, where a retrospective study was done with 111 patients(18). Lofti et al also reported similar findings in a prospective study done which included 258 patients. There was also an association noted between total intraoperative blood loss and infection rate, p = 0.133(p<0.2, CI 80%). Lofti et al conducted a prospective study of 258 patients in order to identify a high risk group for wound infections, there was no association found with relation to intraoperative blood loss or intraoperative blood transfusion(75). There was also a strong association with occurrence of infection and the clinical nodal staging of the disease, with p= 0.184 (p<0.2, CI 80%). This finding was confirmed by Robbins et al who studied 400 cases at the M. D Anderson Cancer Center to determine risk factors in head and neck

patients, their conclusion was that the N stage is predictive of infection. These findings were confirmed further by Lofti et al. Belusic-Gobic did not find any association between N stage of the disease and infection rates.

The N stage disease and the total intraoperative blood loss were associated with increase in wound infection at a confidence interval of 80%, to be statistically significant at 95% the proposed sample size would require completion. Schwartz found that there was an increase in wound infection rate with thrombocytosis (48), which was in contrast to the findings in our study.

In our study age, sex, diabetic status or consumption of tobacco had no bearing on wound infection rates. The dental status in our study had no association on infections which differed from Chaukar et al prospective study of 186 patients(52).

Whether preoperative chemotherapy or radiation would affect the surgical site and cause infections was always debatable. In our study, of the 53 recruited only 1 case had both chemotherapy and radiation preoperatively, there was no association found, as was confirmed by other studies(17,46). Penel in his study showed no association between radiation and wound infection but significance was present for preoperative chemotherapy. Girod et al in a retrospective study of 159 patients found that preoperative radiation therapy significantly increases the post-operative complications(47).

The swabs that were taken from the infected wounds exhibited polymicrobial infection, as was also confirmed by various other studies where there were similar culture growths as shown in the table below:

Author	Organisms cultured
Chaukar et al(52)	*Escherichia coli, Pseudomonas aeruginosa*
Cloke et al(17)	*Staphylococcus sps, streptococcus, pseudomonas, Proteus, Escherichia coli*
Lofti et al(75)	*Pseudomonas, staphylococcus aureus, Escherichia coli, Klebsiella*
Belusic- Gobic et al(18)	*Staphylococcus aureus, Pseudomonas, Actinetobacter, Diphteroides*
Penel et al(19)	*Escherichia coli, Staphylococcus, streptococcus*

Table 18: Studies showing post operative wound cultures.

In our study, out of the total 9 patients who developed wound infection, the organisms that were cultured from the infected wound of 5 patients post operatively were also noted in their preoperative oral cultures. Whether there is a role for preoperative intravenous antibiotics or as local mouthwashes should be strongly considered.

All 53 patients that were recruited for the study had pre-operative oral swabs taken prior to the administration of intravenous antibiotics. Culture growths included in additional to normal oral flora, the numerous pathological organisms that had the potential to cause wound related complications.

Normal oral flora	Pathological organisms with no infection	Pathological organisms with infection	Total oral swabs collected
28 (52.8%)	15 (28.3%)	9 (16.9%)	53(100%)

There were a total of 24 patients who grew pathological organisms in their pre-operative cultures, of these only 9 patients had a post operative wound infection. There were 15 patients who did not have any post operative wound infection, the cause for which can be attributed to either the prophylactic antibiotics or use of triclosan suture coated material intra-operatively. Of the 9 patients who had a surgical site infection post operatively, 50% of the infections were caused by the same organisms that were cultures preoperatively. Whether this calls for further optimization of antibiotic protocols is a cause for further investigations. Studies are currently being formulated in our unit for development of these protocols.

LIMITATIONS:

1. During the duration of the intended study, there was difficulty in procuring the required suture material – Triclosan coated polyglactin suture (Vicryl Plus) from the specific company on time. The net delay led to not being able to complete the sample size by the time of submission of this thesis. The intent is to complete the sample size, so as to provide statistical evidence.
2. There are multiple confounding factors that can cause surgical site infections, only a few of which have been studied in this study. Studying all the factors though important would have been beyond the scope of this study. Further more comprehensive studies have to be carried out to fully understand the complexity of wound infections.

CONCLUSION:

The overall wound infection rate in post operative patients who underwent oral malignancies surgery with the use of triclosan coated polyglactin sutures was 17%. As the clinical and the pathological T staging increased, the incidence of wound infection also increased. This was statistically significant with $p = 0.009$ ($p < 0.05$, confidence interval -95%). There was also statistical significance in relation to total duration of hospital stay and infection with $p=0.02$ ($p < 0.05$, confidence interval -95%). The total duration of antibiotic administration was also significant with those having antibiotics for more than 48hours having a higher risk of infection. Confounding factors were not included as major head and neck dissections received more than 48 hours of antibiotics while wide local excision with primary closure were given only 48 hours of antibiotics. An increase in the risk of infection was also associated with the surgery preformed, the total intra-operative blood loss and the clinical nodal disease prior to surgery. Multivariate analysis was inconclusive and would require completion of the proposed sample size.

The microbiological analysis revealed that the oral microbiology in patients with an oral malignancy is the same as those without. The cultures revealed normal oral flora in addition to pathological organisms such as *Staphylococcus*, *Streptococcus and Klebsiella, Enterobacter*. In wounds that were infected, there

was a similarity in the pre operative culture and cultures taken from the infected site.

This pilot study reveals that post operative wound infection rate in patients with oral malignancy had decreased when triclosan coated sutures were used for surgical closure as compared to those with plain polyglactin 910 sutures, the true statistical significance of the proposed sample size is awaited on completion of the study. Nevertheless, this pilot study reveals that tumour staging of disease is a statistical significant factor to determine post operative surgical site infections.

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ANNEXURES

PROTOCOL

1. Case recruitment, with consent taking from the ward on Monday.
2. ON THE DAY OF SURGERY:

   Prior to administration of antibiotics – 2 oral swab to be taken

   Details regarding- Intraoperative blood loss, lowest intraoperative temperature recording and transfusions to be filled online in operation record.

3. POST- OPERATIVELY:

   Should wound infection occur, 2 oral swabs to be taken.

WOUND INFECTION DEFINED BY CDC CRITERIA INCLUDED IN THE STUDY AS BELOW:

Infection occurs within 30 days post op, must involve at least one of the following:

1. Purulent drainage from the superficial incision.

2. Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.

3. At least one of the following: pain, tenderness, localized swelling, redness, heat *and* superficial incision is deliberately opened by surgeon, *unless* incision is culture-negative.

4. Diagnosis of superficial incisional SSI by the surgeon or attending physician.

Do *not* report the following conditions as SSI:

1. Stitch abscess (minimal inflammation and discharge confined to the points of
2. suture penetration).
3. Incisional SSI that extends into the fascial and muscle layers.

STUDY PROFORMA:

DATE OF RECRUITMENT: _____

SERIAL NO: _____

NAME: _____

HOSPITAL NO: _____

SEX:_____ AGE: _____

HISTORY OF DIABETES MELLITUS: Y/N

IF YES, CONTROLLED OR UNCONTROLLED:

_____

HISTORY OF TOBACCO USAGE:

_____

PREVIOUS HISTORY OF CHEMOTHERAPY:

_____

PREVIOUS HISTORY OF RADIATION

THERAPY:_____

CURRENT DENTAL STATUS (good, bad, edentulous):

_____

PREOPERATIVE INVESTIGATIONS:

HEMOGLOBIN:

_____

TOTAL PLATELETS:

_____

STAGING OF THE DISEASE: Clinical –

_____

Pathological -

_____

SURGERY PROFORMED:

_____

_____

DATE OF SURGERY:

_____

ORAL SWAB TAKEN PREOPERATIVELY ON TABLE: Y/N

TOTAL DURATION OF SURGERY:

_____

TOTAL INTRAOPERATIVE BLOOD LOSS:

_____

LOWEST INTRAOPERATIVE TEMPERATURE

RECORDED:_____

OCCURRENCE OF INFECTION: Y/N

IF YES, SWAB TAKEN: Y/N

TOTAL DURATION OF HOSPITAL

STAY:_____

TOTAL DURATION OF POST OPERATIVE

STAY:_____

ANTIBIOTICS ADMINISTERED:

_____

TOTAL DURATION OF ANTIBIOTIC ADMINSTRATION:

_____

INITIATION OF

RADIATHERAPY:_____

PREOPERATIVE CULTURE GROWTH :

_____

_____

_____

_____

_____

_____

CULTURE GROWTH FROM INFECTED SITE:

_____

_____

_____

_____

_____

_____

INFORMATION SHEET

Thank you for considering to participate in this study. Given here are details pertailing to the study. Should you have any other questions do not hesitate to ask.

WHAT IS THE PURPOSE OF THE STUDY?

Head and neck cancers are very common in our country and those operated are at a risk of developing wound infection. The risk of wound infection is relatively high, 1 in 4 individuals are prone to develop wound infection. The factors that cause wound infection are still unclear. The introduction of perioperative antibiotics has decreased the risk of infections. This study aims to determine whether an antibiotic coated suture will reduce the risk further.

Triclosan sutures have decreased surgical site infections by 30%, but no studies have been done using Triclosan sutures in oral malignancy – thus this study.

WHAT IS TRICLOSAN?

Tricolsan is an antimicrobial compound that acts by preventing reproduction of microorganisms. It was introduced initially into soaps and detergents as an antibacterial substance which prevents local colonization.

HOW DOES IT WORK?

When it is incorporated into sutures, Triclosan prevents bacterial colonization of the suture line and the surrounding area where there is local absorption of the antimicrobial into the tissue.

ANY ADVERSE EFFECTS OF USING TRICLOSAN?

Studies have not shown any evidence of skin sensitisation, or any other adverse effects and no difference in intraoperative handling by surgeons.

WHAT IS MY ROLE AS A PATIENT?

The role you play by agreeing to participate in this study is by ensuring to follow up in our hospital for radiotherapy in those where it is indicated or otherwise a period of 6 weeks.

HOW DOES THE STUDY PROCEED?

Once the participant has agreed to be part of the trial and the written consult is given there will be the standard pre- operative blood investigations done.

Intraoperatively, the participant will receive the first dose of antibiotics which will be continued for the total of 5 days postoperatively as is the unit policy. Postoperatively

should a participant get a wound infection, microbiological samples will be taken and they will be treated for the same.

WILL THERE ANY ADDED EXPENSES?

All expenses conferred in this study – additional cost of suture materials will be covered by external funding. Should a wound infection occur, the expenses of the bacterial culture will be covered by these external funds.

No additional expenses will be imposed on the participant.

WHAT HAPPENS IF THERE IS AN UNTOWARD EVENT?

If there is any untoward event – that is an allergic reaction to suture material or any other unprecedented reaction, you can freely withdraw from the study and expenses will covered by unit fund.

Wound infection, sepsis is not considered as an untoward event.

In case of any queries, please contact :

Dr. Abhilasha Singh,

CONSENT FORMS

Informed Consent form to participate in a research study

Study Title: Use of triclosan coated polyglactin 910 sutures versus plain polyglactin 910 sutures in oral malignancy.

Study Number: _____

Subject's Initials: _____ Subject's Name: _____

Date of Birth / Age: _____

(Subject)

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions.

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published.

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

(v) I agree to take part in the above study.

Signature/ Thumb impression of the Participant :

Date: _____/_____/_____

Signatory's Name: _____ Signature:

Signature of the Investigator: Signature of the Witness:

Date: _____/_____/_____ Date: _____/_____/_____

Study Investigator's Name: _____ Name & Address of the Witness

In case of any queries, please contact :

Dr. Abhilasha Singh,

Master Data Sheet

SERIAL NO.	SEX	AGE	DM	DM STATUS	TOBACCO	CHEMO	RT	DENTAL STATUS	HB	PLATELET	CLI - T	CLI - N	PATH - T	PATH - N	SURGERY	DURATION	BLOOD LOSS	HOSPITAL STAY	POST OP STAY	DURATION OF ABX	STARTING RT	PATHOLOGY	INFECTION
1	1	2	2	0	1	2	2	2	2	0	2	5	4	1	2	2	2	2	2	2	2	1	1
2	1	2	2	0	1	2	2	1	2	0	2	5	1	5	1	1	2	2	2	2	0	1	2
3	1	2	1	1	1	2	2	2	2	0	1	5	1	5	1			1	1	1	0	1	2
4	1	2	2	0	2	2	2	1	2	1	1	5	5	5	1	1	1	1	1	1	0	0	2
5	2	2	2	0	2	2	2	3	2	0	2	5	5	5	1	2	1	1	2	1	0	0	2
6	1	2	2	0	1	2	2	1	2	1	2	5	1	5	1	1	1	2	2	2	2	0	2
7	1	1	2	0	2	2	2	1	2	0	1	5	1	5	2	2	2	1	2	1	0	2	2
8	2	2	2	0	1	2	1	2	2	1	2	1	1	5	2	2	2	2	2	2	0	1	2
9	2	2	2	0	1	2	2	2	2	0	4	1	4	2	2	2	2	2	2	2	1	1	2
10	1	1	2	0	2	1	2	1	2	2	2	5	1	5	1	1	1	2	2	2	1	2	2
11	2	1	2	0	1	2	2	2	2	0	1	5	2	5	1	2	1	1	1	1	2	1	2
12	2	2	2	0	1	2	2	2	2	1	4	2	1	2	2	2	2	1	2	2	2	1	2
13	2	2	2	0	1	2	2	2	2	0	4	1	2	5	2	2	2	2	2	2	1	1	1
14	1	2	2	0	2	2	2	1	2	1	4	5	2	5	2	2	2	2	2	2	2	1	2
15	2	2	2	0	1	2	2	2	2	0	2	5	2	2	1	2	2	1	2	2	0	1	2
16	1	2	2	0	1	2	2	1	2	1	4	1	4	2	2	2	2	2	2	1	1	1	2
17	1	2	2	0	1	2	2	1	2	1	2	5	1	5	1	2	1	1	2	2	2	1	2
19	2	2	2	0	1	2	2	2	2	1	2	1	1	1	1	2	1	2	1	1	1	1	2
20	1	2	2	0	2	2	2	1	2	1	4	2	2	5	2	2	2	2	2	2	1	1	2
21	2	2	2	0	1	2	2	3	2	1	0	2	1	1	1	2	2	2	2	2	1	1	2
22	2	2	1	2	2	2	1	2	2	1	4	2	5	1	2	2	2	1	2	2	0	1	2
23	2	2	2	0	1	2	2	2	1	1	3	1	4	5	2	2	2	2	2	2	0	1	2
24	1	2	2	0	2	2	2	1	2	1	2	5	4	5	1	2	2	2	2	2	0	1	2
25	2	2	2	0	1	2	2	2	2	0	2	5	1	5	1	2	2	1	1	1	0	2	2
26	1	2	2	0	2	2	2	2	2	0	1	5	1	5	1	1	1	1	1	1	0	2	2
27	2	2	1	2	1	1	2	2	2	1	4	3	4	2	2	2	2	2	2	2	0	1	2
28	2	1	2	0	2	2	2	2	2	1	3	2	3	0	2	2	2	2	2	2	2	1	1
29	1	2	2	0	1	2	2	2	2	0	1	5	2	5	1	2	1	1	1	1	0	1	2

Master Data Sheet

SERIAL NO.	SEX	AGE	DM	DM STATUS	TOBACCO	CHEMO	RT	DENTAL STATUS	HB	PLATELET	CLI - T	CLI - N	PATH - T	PATH - N	SURGERY	DURATION	BLOOD LOSS	HOSPITAL STAY	POST OP STAY	DURATION OF ABX	STARTING RT	PATHOLOGY	INFECTION
30	2	1	2	0	1	2	2	2	2	1	2	5	1	3	1	1	1	1	2	1	0	1	2
31	1	1	2	0	1	2	2	2	2	0	1	5	1	2	1	1	1	1	1	1	0	1	2
32	2	2	2	0	1	2	2	1	2	2	4	2	4	2	2	2	2	2	2	2	0	1	1
33	1	1	2	0	1	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	0	1	2
34	1	1	2	0	1	2	2	1	2	1	2	5	1	1	1	2	2	1	1	2	0	1	2
35	2	2	2	0	2	2	2	2	2	0	4	5	2	5	2	2	2	2	2	2	2	1	1
36	1	1	2	0	2	2	2	1	2	1	1	5	5	5	1	1	1	1	2	2	0	1	2
37	1	2	2	0	1	2	2	2	2	1	2	1	4	5	2	2	2	2	2	2	0	1	2
38	2	2	2	0	1	2	2	3	2	0	2	5	2	5	2	2	2	2	2	1	0	1	2
39	1	2	2	0	1	2	2	2	2	1	1	5	1	5	1	2	2	1	1	1	0	1	2
40	1	2	2	0	1	2	2	2	2	0	4	2	3	2	2	2	2	2	2	2	1	1	1
41	1	2	1	2	1	2	2	2	2	1	4	2	4	2	2	2	1	2	2	2	0	1	2
42	1	2	2	0	2	2	2	1	2	1	1	5	1	5	1	1	1	2	2	2	0	1	1
43	2	2	2	0	2	2	2	1	2	0	2	5	2	5	2	1	1	2	2	2	0	1	2
44	2	1	2	0	2	2	2	1	2	0	1	5	1	5	2	2	2	2	2	2	0	1	2
45	1	2	2	0	1	2	2	2	2	0	2	5	1	2	2	2	2	2	2	2	1	1	2
46	1	2	2	0	2	2	2	1	2	0	2	2	2	1	2	2	2	2	2	1	0	1	2
47	1	2	2	0	2	2	2	1	2	1	2	5	4	1	1	1	1	2	2	1	1	1	2
48	2	2	1	2	2	2	2	2	2	0	3	2	4	1	2	2	2	2	2	2	1	1	1
49	1	1	2	0	1	2	2	2	2	0	2	1	2	5	2	2	2	2	2	1	0	1	2
50	2	2	2	0	2	1	1	3	2	0	1	5	1	0	2	2	1	2	2	1	0	1	2
51	1	2	2	0	2	2	2	1	2	1	4	2	3	5	2	2	2	2	2	2	2	1	1
52	1	2	2	0	2	2	2	2	2	1	2	5	1	5	1	2	1	1	2	1	0	1	2
53	1	2	2	0	1	2	2	2	2	1	3	2	2	2	2	2	2	2	2	1	1	1	2
54	1	2	2	0	1	2	2	2	2	1	4	2	4	1	2	2	2	2	2	2	1	1	2

ABSTRACT:

OBJECTIVES:

To study post operative wound infection rates in patients with oral malignancy following use of antimicrobial coated polyglactin 910 sutures as compared to plain polyglactin 910 sutures.

METHODS: A pilot study of consecutive cases that underwent surgery for oral malignancies with all surgical sites being closed with triclosan coated polyglactin 910 sutures from 1st January 2014 to 31st July 2015. A total number of 53 patients were recruited and oral swabs was taken and sent for microbiological analysis prior to antibiotic administration. The overall wound infection rate was then compared retrospectively to the infection rate in the previous year in patients where the surgical sites sutured with plain polyglactin 910. Independent variables were analyzed by Chi-Squared test; multiple logistic regression was preformed to account for multiple risk factors.

RESULTS & CONCLUSIONS: The overall wound infection rate in post operative patients who underwent oral malignancies surgery with triclosan coated polyglactin sutures was 17%. The wound infection rate had decreased from 23.86% when plain polyglactin sutures were used. Multivariate analysis was inconclusive and requires completion of the proposed sample size.

The microbiological analysis revealed that the oral microbiology in patients with an oral malignancy is the same as those without.

Nevertheless, this pilot study reveals that tumour staging of disease is a statistical significant factor to determine post operative surgical site infections.

KEYWORDS : Triclosan, wound infection, oral malignancy